John Fry

# Presenting Symptoms in Childhood

Butterworths

Written by a family doctor, this book concerns the symptoms of children's diseases and deals with their causes, diagnosis, pathology and significance and is based on the author's experience in general practice. It is the common symptoms which are met with almost daily that are considered and the more common specific diseases that are discussed.

Infections, particularly of the respiratory and digestive tracts, account for a major proportion of all children's diseases and the greater number of chapters are devoted to these two systems. The author presents a diagnostic approach to children's diseases and the book will be of special interest to general practitioners, senior medical students and to all those who are concerned with illness in childhood.

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# PRESENTING SYMPTOMS IN CHILDHOOD

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# INTRODUCTION

This book presents a diagnostic approach to children's diseases. It is concerned with *symptoms*, because as a general rule it is a symptom that the mother has recognized that brings the child to the doctor.

Starting with the symptom, its causes, pathology, diagnosis and significance are discussed and the important specific conditions associated with the particular symptom are briefly described.

Certain features have been duplicated in the text and the author makes no apology for this. It has been done deliberately so that the reader will not have to refer too often to other sections.

It is based on experience in general practice and therefore it is the common symptoms which the family doctor meets almost daily that are considered, and it is the more common specific diseases that are discussed.

To keep the situation in perspective it is useful to see what are *the common diseases of children.*

*Disease in the community*—Infective illnesses account for the great majority (around 75 per cent) of all sickness in children at home. Three groups—respiratory and gastro-intestinal infections and the common fevers—make up most of these infections. Symptoms such as cough, wheezy chest, sore throat, earache, vomiting, diarrhoea and rashes are the usual presentations. Accidents and sickness related to environment or development account for the majority of non-infective illnesses.

In most of these situations the mother is able to cope herself and will need to seek her family doctor's advice only in about a third or a quarter of all her children's illnesses.

*Disease in general practice*—In general practice the common children's illnesses follow a similar pattern. Respiratory infections with colds, coughs, earaches and sore throats account for a third to a half of all the diseases seen by the practitioner. The common fevers, skin disorders and gastro-intestinal troubles account for about a tenth each. Emotional problems are another important group, not so much because they present as such but because they are involved in many other conditions; in assessing the significance of any symptom the underlying emotional problems of both mother and child must receive due consideration.

*Disease in hospital*—The most frequent reasons for admitting children to hospital are as follows (expressed as percentages of all children's admissions): Removal of tonsils and adenoids (23 per cent); respiratory infections (11 per cent); other infections (poliomyelitis, tuberculosis, gastro-intestinal infections) (10 per cent); accidents (10 per cent); congenital malformation (6 per cent); appendicitis (5 per cent); skin, joint and bone disorders (5 per cent); ear infections and disorders of central nervous system (4 per cent); squint and eye disorders (4 per cent); circumcision (2 per cent); asthma and allergy (2 per cent); mental disorders (0·5 per cent). These are the Ministry of Health figures for 1955 and 1956.

Here again the importance of respiratory and other infections as causes of illnesses is demonstrated.

*Causes of death*—In 1959 there were just over 20,000 deaths in children under 10 years of age. Although some were preventable others must have been inevitable because of congenital and other defects which made life impossible.

The seven most frequent causes of death in 1959, in order, were infants' diseases, congenital defects, respiratory infections, accidents, digestive disorders, general infections and malignant new growths.

From this brief review it is apparent from all sources that infections, particularly of the respiratory and digestive tracts, account for a major proportion of all children's illnesses and it is with these two systems that the largest part of this book is concerned.

In stressing that 'common conditions commonly occur' it is nevertheless important always to consider the possibility of more serious though rare conditions that may require early diagnosis if they are to be managed at a curable stage.

JOHN FRY

*Beckenham, Kent*, 1962

# ABDOMINAL PAIN

Abdominal pain is so common that at one time or another most children complain of pains in the tummy. Although in the majority of instances the symptom will have no serious significance it must always be taken seriously so that a surgical emergency is not missed, and care must be taken to differentiate between local disease and the pain as a manifestation of more general conditions.

## CAUSES

### ACUTE

*Intra-abdominal*

The main acute causes of intra-abdominal pain are as follows.

(1) Underfeeding, which will lead to abdominal pains, irritability and failure to thrive.

(2) Inflammation: acute gastro-enteritis or food poisoning, appendicitis, mesenteric adenitis.

(3) Obstruction: intussusception, bands and adhesions, strangulated hernia, malrotation of gut and volvulus.

(4) Urinary causes: pyelitis, renal colic and hydronephrosis.

*Extra-abdominal*

Usual acute causes of extra-abdominal pain are epidemic myalgia (Bornholm disease), pneumonia and acute otitis media. Rare causes are meningitis, rheumatic fever, Henoch–Schönlein (anaphylactoid) purpura, diabetic ketosis and porphyria.

### CHRONIC AND RECURRENT

The chronic and recurrent causes of intra-abdominal pain may be listed as:

*Intra-abdominal*

(1) Infancy: 'colic' and 'wind'.

(2) Obstruction: constipation, polyp or neoplasm, volvulus (congenital).

(3) Colics: renal, or, more rarely, biliary or pancreatic.

(4) Inflammatory: appendicitis (recurrent), mesenteric adenitis.

(5) Gynaecological: ovulation, dysmenorrhoea, torsion of ovarian cyst (rare).

(6) Peptic ulcer.

(7) Renal disorders: infections or congenital abnormalities.

*Extra-abdominal*

The more usual chronic and recurrent causes of extra-abdominal pain are 'periodic syndrome', emotional disturbances ('stress') and epilepsy. Rarer causes include spinal disease (tuberculosis, tumour), lead poisoning and porphyria.

## PATHOLOGY

The pathological reactions depend on the causes.

## DIAGNOSIS

Although in the majority of children with abdominal pains no serious causes are ever found, the situation must always be considered as one of a possible serious organic disorder until proved otherwise.

*Recognition of Abdominal Pain*

The first step in diagnosis must be to decide whether abdominal pain is present.

In infancy pain from any source will cause screaming and drawing-up of the child's legs and it is not easy to decide when the pain is due to a cause within the abdomen. The mother's history is of the greatest importance and will give information as to whether the disturbance is constant or intermittent, whether there are associated vomiting or bowel upsets, fever or other points. All these features help to localize and establish the diagnosis.

In early childhood, up to the age of 7 or 8 years, most abdominal pains are localized by the child to the umbilicus.

In some children 'tummy ache' is the presenting complaint of all illnesses, whatever their causes.

Older children, over 8 years, are excellent witnesses, especially when examined away from their mother. They should be treated as young adults and their stories should be accepted with few reservations.

In no other situation is it so important to consider the child with

abdominal pain as a whole. Examination and the history must cover all systems and never be limited to the abdomen. The chest, throat, ears and urine must all be examined and particular attention paid to the child's general health and behaviour. Emotional relationships between the child and mother and with other relatives and friends must also be considered in making an assessment.

*Diagnostic Approach*

The mother should be seen first and a history taken. Inquiries must be made on the periodicity, severity, site and character of the pain. The presence of vomiting, fever and alterations in bowel behaviour must be noted and related to the abdominal pain. Patient examination is then required to assess the significance of the child's posture and appearance, of any tenderness, guarding and abdominal distension. Feeding habits are of importance in infants. A common cause of abdominal pain is underfeeding because of the mother's fear of overfeeding.

*Associated symptoms*—These are probably more significant than abdominal signs. Vomiting when present should make the doctor very wary and if repeated without diarrhoea the probability of some serious organic cause must be considered. Fever in children is not of much significance but it obviously implies a general disturbance the cause of which must be explored. Bowel disturbances are frequent but should not of themselves be too readily put forward as the cause of pain. Constipation is not a frequent cause of acute abdominal pain. It is highly dangerous to ascribe 'constipation' as the cause of abdominal pain and to treat the child with laxatives. It is the surest way of causing an inflamed appendix to perforate or an obstructed bowel to rupture. Diarrhoea accompanying abdominal pain may not be the result of dietetic indiscretions or 'enteritis'. It may be due to 'pelvic' appendicitis or a pneumonia.

*Examination*

One should never rush at the child and start palpating the abdomen. This will only create fear and resistance. Examination should follow the history, during which the doctor should make friends with his patient. The chest should be examined first and then the painful abdomen. Needless to say, gentleness is essential, but over-persistence is useless in the face of uncooperation. A diagnosis may have to be made without a satisfactory abdominal palpation.

A rectal examination must always be done and the urine examined for albumin, sugar and pus.

## SIGNIFICANCE

The child with 'tummy ache' presents the doctor with a number of distinct problems of diagnosis and management.

The doctor has first to try to make a diagnosis, and this is by no means easy. Studies have shown that a large proportion of these aches belong to rather indefinite and indefinable groups which tend to be self-limiting and recurrent and of little serious significance. Having made a tentative diagnosis, the doctor has next to decide whether he is dealing with a 'serious' disorder or not. If 'serious' he next has to consider whether the child should be 'observed' and treated at home or admitted to hospital. It is more important for the family doctor to suspect quickly the presence of an 'acute abdomen' than to be correct after some delay with the final diagnostic label.

More often the doctor is faced with the problem of a child with recurrent abdominal pain. At the time when the child is seen and examined between attacks there are generally no abnormal signs. Any decision as to the underlying cause and on the correct management has to be based on a careful analysis of the mother's history together with any ancillary investigations that may be considered necessary.

## SPECIFIC CONDITIONS

### THE 'PERIODIC SYNDROME'

*Aetiology*

Recurrent bouts of abdominal pain are frequent in children and are often associated with vomiting, pallor, headache, fever and limb pains. The label of 'periodic syndrome' is given to such a collection of symptoms and signs but in spite of this apparent specificity we are still not quite certain of the exact causes.

*Incidence*—More than a tenth of all children suffer from recurrent abdominal pains that fit into this syndrome. Girls are affected more often than boys.

*Age*—Some children begin to suffer attacks in infancy which may persist throughout childhood. More usually the attacks begin in the pre-school years and are particularly frequent in girls between 9 and 12 years of age. In many children with this syndrome there is a family history of similar pains in relatives and of peptic ulcer, headaches and nervous breakdowns.

*Clinical Features*

Abdominal pain is the main feature. It is localized to the umbilicus and is described as a dull pain 'like wanting to be excused'. It occurs at all times, but is particularly frequent in the mornings in children at school. As a rule the pains are of short duration, lasting only a few minutes, and recur at quite irregular intervals. Vomiting may occur in association with the pain and generally precedes the pain. Headache may be a feature, fever is present in a minority in whom attacks tend to persist for a day or more. Pallor is a significant sign often noted by the mother.

*Course*—Attacks of abdominal pain in these children reach a peak between 5 and 7 years of age in boys, and between 8 and 12 years in girls. After these periods they tend to become less frequent and less severe. In a few attacks may persist or be replaced in adult life by migrainous headaches or other psychosomatic conditions.

### UNDERFEEDING

In infancy hunger is a most frequent cause of recurrent abdominal pain. The infant is usually a first baby. The mother is inexperienced and keen. She has probably read books and listened to the advice of her elders and is terrified of 'overfeeding'. She consequently goes by the book and keeps to a fixed timetable. The baby cries a lot and draws up his legs and appears in pain. Examination reveals an irritable but healthy child.

The diagnosis is confirmed, and the condition cured, by giving more food and by reassuring the mother that it is really impossible to overfeed her baby.

### GASTRO-ENTERITIS

A mild form of acute gastro-intestinal infection, probably viral, with vomiting and diarrhoea is a frequent cause of sudden abdominal pain in children. Often occurring in epidemic form with more than one case in the family, it comes on suddenly with repeated vomiting followed by bouts of colicky abdominal pain and then a few hours later by diarrhoea. The whole attack lasts about 24–48 hours.

There is little constitutional upset and abdominal examination does not reveal any abnormal signs.

This short and sharp condition with few serious effects is quite different from the rare and severe gastro-enteritis that affects infants with many systemic complications.

## ACUTE APPENDICITIS

Although rare below the age of 3 years, acute appendicitis is a frequent acute abdominal emergency in children. At all ages any abdominal pain that has continued without intermission for several hours must be regarded as possible appendicitis, especially if it is associated with vomiting. It is well, however, to keep the situation in perspective and to realize that while it is a frequent condition in a children's ward in hospital the average general practitioner may only come across one new case of acute appendicitis in children in a year.

*Clinical Features*

*In children over 3 years of age*—As a general rule there is no preceding history of recurrent abdominal pains. The onset is sudden and can often be timed to the minute by the mother. Pain is the main symptom. It is generally continuous. It is not as severe as the pain of renal or biliary colic or of an intussusception. It may be paroxysmal, the child being able to play and run about between the attacks of pain. Localization is poor in children and it is unusual to get the story of pain starting in the epigastrium and shifting to the right iliac fossa. Much more often the child has a 'tummy ache around the middle'.

Vomiting occurs in almost all cases once or twice. It is only rarely persistent. Bowels may not be opened or alternatively there may be diarrhoea when the appendix is situated in the pelvis.

The child looks ill. He lies still, either on his back or on his right side, with the knees bent. Movement is resented.

The tongue is coated and the breath smells. Gentle palpation will reveal tenderness in the lower abdomen most marked on the right and the child will actively push away the examiner's hand.

Later in the disease the child becomes shocked, dehydrated and grey in colour with a rapid, thready pulse. Tenderness becomes generalized and vomiting repeated.

A rectal examination is essential if the diagnosis is in doubt, especially in the case of a pelvic appendix when symptoms and signs are equivocal (little abdominal tenderness and diarrhoea); in this case there is marked tenderness on rectal examination.

*In infants under 3 years of age*—The diagnosis is often extremely difficult. At the same time the situation is much more dangerous, with a tendency to early perforation and generalized peritonitis. Early and accurate diagnosis is very important.

The initial features are general in character: malaise, peevishness,

irritability or lassitude, refusal of food, vomiting and a fever often up to 104°F. (It used to be said that if the temperature was over 101°F appendicitis was unlikely. This is a very dangerous fallacy.)

Pain is a very indefinite affair in these infants, but the mother is able to tell that her child is in pain and ill. Vomiting is invariable and repeated. Diarrhoea is much more common than in older children. This association with diarrhoea and vomiting may lead to the dangerous erroneous labels of 'food upset' or 'gastro-enteritis'.

### APPENDIX ABSCESS

Sometimes the doctor is not called until the condition has been present for some days or perhaps the early diagnosis has been missed. The story is then of an acute or subacute illness over some days with continued abdominal pains, general malaise and fever.

On examination a tender mass will be felt either in the lower abdomen or in the pelvis on rectal examination.

### MESENTERIC ADENITIS

This is a condition that is said to be much more frequent than appendicitis, yet it is a much more indefinite diagnosis.

*Clinical Features*

Pain is the presenting symptom. It is central and intermittent in character. Attacks last up to 15 minutes and are severe. Between the attacks the child is active and happy. Vomiting occurs in severe attacks but is not so frequent as in appendicitis. Bowels are not often disturbed. The tongue is clean. Tenderness is neither marked nor localized. The attacks are self-limited and recurrences not unusual. The condition often follows an acute upper respiratory infection.

### INTUSSUSCEPTION

This causes partial or complete intestinal obstruction due to invagination of a proximal portion of gut into the more distal portion. It is most common around 9 months of age, but can occur between 2 months and 2 years of age. Boys are affected much more often than girls.

*Clinical Features*

The onset is sudden and may be associated with an acute respiratory infection. The exact time can often be given by the mother.

Pain is severe and intermittent and may be followed by pallor and collapse. The spasm, with screaming and drawing-up of the

knees or whimpering and whining, recurs at increasingly frequent intervals and the child becomes shocked and collapsed. Vomiting occurs but is not a prominent feature.

One or two normal bowel motions may be passed at the onset. Blood mixed with mucus ('red currant jelly') is pathognomonic when present, especially following rectal examination.

During the attacks of pain the abdomen is rigid and tender but between the attacks it may be quite soft. The characteristic sausage-shaped tumour may be felt and there is an 'emptiness' in right iliac fossa.

The *diagnosis* should be suspected on the history of intermittent pain, collapse and vomiting, and one must never wait to feel the 'tumour' or to see 'red currant jelly' stools.

### ACUTE INTESTINAL OBSTRUCTION

Acute intestinal obstruction occurs most often at two distinct periods —in newborn infants and in older children between 2 and 5 years of age. In older children the classical features of intermittent colicky abdominal pain, copious vomiting, absolute constipation and abdominal distension are present together with visible peristalsis and loud bowel sounds. The most frequent causes are bands or adhesions, intussusception, strangulated hernia or malrotated gut and volvulus.

In newborn infants vomiting is the outstanding feature. This starts on the first or second day and persists. The baby also appears to suffer bouts of colicky pain. Abdominal distension is associated with obstructions in the lower parts of the small intestines or large bowel but is not prominent in high duodenal obstructions.

### ACUTE URINARY INFECTIONS

Acute urinary infections are not uncommon in young children. Much more frequent in girls than boys, they can occur at any age but especially in infants and children under 5 years of age.

In infants the presentation is that of an acute onset with general malaise and fever. The child is flushed and ill. There is often vomiting and sometimes diarrhoea. Convulsions may occur. Abdominal pain is not a marked feature.

In older children in addition to the general disturbance with fever and malaise there will be frequency and dysuria. Children under 5 years rarely complain of urinary discomfort and the diagnosis will only be made by testing the urine for pus cells. The pain is generally

continuous with occasional colicky attacks. Localization is poor and the site of the pain tends to be referred to the central abdomen.

## EXTRA-ABDOMINAL CAUSES

*Pneumonia*

In pneumonia upper abdominal pain may be an early symptom. The child is restless, with high fever, rapid respirations and movements of the nostrils. Abnormal signs in the chest such as rhonchi and rales may be present, but in the early stages there may be no abnormalities in the chest. Delirium and shivering, often present in pneumonia, never occur in appendicitis with which diagnostic confusion may occur.

*Acute Otitis Media*

Acute otitis media in infants may present with fever, irritability and spasms of pain that may be referred to the abdomen. It is important therefore to examine routinely the ears of all children.

*Henoch–Schönlein Purpura*

Henoch–Schönlein (anaphylactoid) purpura is a rare condition in practice (about one new case for every 10 years in general practice). Abdominal pain may occur some time before the characteristic rash, a mixture of urticaria and purpura, appears on the elbows, buttocks and legs. There may be rectal bleeding and there is often renal involvement with microscopical haematuria and albuminuria.

*Epidemic Myalgia (Bornholm Disease)*

This epidemic disorder, which also occurs sporadically, is most prevalent in children between 5 and 15 years of age. It is a virus infection caused by the Coxsackie group of entero-viruses, and is a dramatic but benign illness.

The onset is sudden. Pain is characteristically situated in the upper abdomen and has been described as 'an iron band around the top part of the stomach'.

It is made worse on breathing, coughing and sneezing. There is often a curious expiratory grunt. A feature of the pain is to improve and then recur a few hours or days later. Associated symptoms are headache, sweating, nausea and vomiting, but there is little systemic upset. A loud rub on respiration may be heard over the lower chest.

The course of the disease is for a spontaneous recovery within a few days (but it may take a couple of weeks), without any serious complications.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Acute intra-abdominal pain | | | | |
| (1) Underfeeding | First baby as a rule: anxious mother who is afraid of overfeeding; baby cries and draws up knees Constipation | Clinical | Advice and increase in feeding | Good |
| (2) Inflammation | Acute appendicitis: rare under 3 years, increasingly common after this age Gastro-enteritis: frequent cause of colic and diarrhoea Mesenteric adenitis: indefinite condition | Clinical | Surgery for appendicitis | Good |
| (3) Obstruction | Neonatal causes and intussusception most frequent causes Abdominal pain accompanied by vomiting and constipation | (1) Clinical (2) Radiograph of abdomen | Surgery | Depends on cause |
| (4) Urinary causes | Pyelitis: frequent in girls Urinary symptoms usually present High fever | (1) Clinical (2) Urine | (1) Antibiotics (2) Investigate fully in recurrent cases | Good usually, unless underlying abnormalities |
| Acute extra-abdominal pain | Abdominal pain may be a feature of epidemic myalgia, pneumonia, acute otitis media and *rarely* of meningitis, rheumatic fever, diabetic ketosis, Henoch–Schönlein purpura and porphyria | That of specific condition | That of specific condition | |
| Chronic recurrent intra-abdominal pain | | | | |
| (1) Wind and colic in infants | Recurrent attacks of acute abdominal pain; healthy baby; relieved by passage of wind | Clinical | Advice | Good |
| (2) Obstruction | Rare—Abdominal pain is unusual in simple constipation; organic obstruction should be considered | (1) Clinical (2) Radiograph | Depends on cause | Depends on cause |
| (3) Colics (i.e. renal, biliary or pancreatic) | Rare in children | That of specific condition | That of specific condition | Depends on cause |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| (4) Inflammatory | Recurrent attacks of subacute appendicitis or mesenteric adenitis | Clinical | Surgery if indicated | Good |
| (5) Gynaecological | In girls between 10 and 15 years; gynaecological conditions may cause recurrent abdominal pains | Clinical | That of primary condition | Good |
| (6) Peptic ulcer | Rare—Consider in recurrent pain associated with meals | (1) Clinical<br>(2) Radiograph | Medical regimen | May require surgery later |
| (7) Renal disorders | Recurrent attacks of abdominal pain may occur in congenital abnormalities | (1) Clinical<br>(2) Urine<br>(3) Radiograph | Depends on cause | Depends on cause |
| Chronic recurrent extra-abdominal pain | | | | |
| (1) Periodic syndrome | Frequent cause of recurrent abdominal pain; closely related to emotional problems | Clinical | Discussion of emotional problems | Good |
| (2) Rare causes | Spinal disease, porphyria, lead poisoning and epilepsy | Depends on primary condition | Depends on primary condition | Depends on primary condition |

# ABDOMINAL SWELLING

Swelling of the abdomen not infrequently causes the mother to bring her child to her family doctor. In the majority of instances the cause is physiological and reassurance is all that is required, but an appreciation of other more serious possibilities is necessary.

## CAUSES

The causes of abdominal swelling may be subdivided as follows.

(1) Physiological. Until the age of about 4 or 5 years the normal child's abdomen tends to be rather protuberant. This is the most common 'cause' of swollen abdomen.

(2) Fluid, which may be grouped under two headings:

(a) Generalized: Chronic peritonitis—tuberculous; acute peritonitis—bacterial; heart failure—congenital defects, rheumatic heart disease; renal failure—nephritis, nephrotic syndrome; liver disease—cirrhosis, thrombosis of portal vein or inferior vena cava; malignant disease—peritoneal deposits; nutritional oedema —hypoproteinaemia; polyserositis; lymphatic obstruction—chylous ascites.

(b) Localized: appendix abscess; encysted ascites—usually tuberculous peritonitis; cysts—ovarian, omental, mesenteric, pancreatic.

(3) Gaseous, comprising chronic constipation, Hirschsprung's disease, idiopathic megacolon, intestinal obstruction, coeliac disease, gargoylism, rickets, cretinism.

(4) Solid masses, which may be:

(a) Renal—hydronephrosis; polycystic kidneys, Wilms' tumour.

(b) Suprarenal—neuroblastoma of medulla.

(c) Of the spleen—lipoidoses, e.g. Gaucher's disease, gargoylism, acholuric jaundice, Banti's disease, leukaemia; Hodgkin's disease; malaria; kala-azar; glandular fever.

(d) Of the liver—cirrhosis; malignant disease, either primary or secondary, e.g. suprarenal; leukaemia; Hodgkin's disease; von Gierke's disease.

(e) Of the gut—faeces; Crohn's disease; appendix abscess.

(f) Peritoneal—retroperitoneal sarcoma, lymphosarcoma;

tuberculous glands; psoas abscess (from tuberculosis of lumbar spine); Hodgkin's disease.

(5) Abdominal wall. Here abdominal swelling may be caused by:

(a) Hernia—umbilical; epigastric.

(b) Neoplasms—lipoma; Hodgkin's disease.

(c) Inflammation—acute cellulitis.

## PATHOLOGY

The pathology of the various conditions depends on the individual disorder.

## DIAGNOSIS

Our first concern is to decide whether the swelling is a normal physiological condition or whether it is the result of some serious intra-abdominal abnormality.

The child is always generally unwell when there is some pathological cause present. In the normal physiological enlargement, apart from the protuberance, which disappears completely on lying down, the child is quite healthy. On the other hand, where there is some serious cause the child is not thriving. He looks unwell on first inspection and on further questioning there are obvious associated symptoms of the primary condition.

Examination of the abdomen (but the other systems must also always be examined) is directed towards deciding the cause of the enlargement. Is it due to fluid accumulation? To gaseous distension? Or to enlargement of a solid viscus?

Ascites should be diagnosed by the fluid thrill, the shifting dullness and by the generalized and uniform swelling of the abdomen. Gaseous distension also results in a uniform enlargement but often coils of distended gut can be seen with active peristalsis and the percussion note is very tympanitic with no dullness, shifting or otherwise. The clinical features of the solid masses depend on the viscus involved. It is generally easy to palpate the liver when enlarged, but it is often very difficult to differentiate between the enlarged spleen and kidney. The spleen tends to be more anterior and it is usually possible to get behind it. The kidney, lying more posterior, presents in the loin and can be felt bimanually. If both kidneys are enlarged then the cause is likely to be either polycystic disease or bilateral hydronephrosis from a congenital bladder-neck obstruction. A retroperitoneal sarcoma is generally a fixed mass in the midline in a very sick child.

The most useful investigations are the intravenous pyelogram, which will demonstrate the kidneys and suprarenal areas—frequent sites of malignancies in infants, and a full blood count.

## SIGNIFICANCE

Swelling of the abdomen is a symptom or a sign of some disorder. The functional disturbance that occurs must therefore be related to the primary cause. The swelling itself is a discomfort for the child and an anxiety for the mother.

The most frequent 'cause' is a physiological 'enlargement' resulting from the fact that the young child's abdominal contents, especially the liver and the small gut, are relatively overlarge for the abdomen, which is pushed out when the child stands. This protuberance disappears when the stage of rapid growth is reached, after 5 years of age.

The next most frequent cause is chronic constipation. In infants about one-third of all solid masses in the abdomen are malignant. In older children this proportion is much less and the great majority are benign. It is important to decide as soon as possible whether or not the mass is malignant, and if a conclusive diagnosis cannot be made clinically or with x-rays and pathological tests then an early laparotomy should be carried out to determine the nature of the swelling.

## TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Physiological | Protuberant abdomen due to relative physiological enlargement of abdominal contents | Healthy child with no clinical abnormalities | Reassurance | Normal shape at around 5 years of age |
| Fluid | Ascites may be due to acute or chronic peritonitis; heart failure; | (1) Clinical features of ascites | That of specific condition | Generally poor but depends on primary disease |
| (a) Generalized | Renal failure; liver disease; malignant disease; nutritional oedema; polyserositis; lymphatic obstruction | (2) Suggestive signs of primary disease | | |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| (b) Localized | Appendix abscess—Following acute onset with abdominal pain and vomiting, inflammation localizes with mass in right iliac fossa | (1) Clinical<br>(2) High white cell count | Surgery after localization | Good |
| | Encysted ascites—Rare feature of tuberculous peritonitis | (1) Clinical<br>(2) Tuberculin test | That of specific condition | Good with specific treatemnt |
| | Cysts—Ovarian, mesenteric, omental or pancreatic | Clinical | Surgery | Good |
| Gaseous | Chronic constipation; Hirschsprung's disease; idiopathic megacolon; intestinal obstruction; coeliac disease; gargoylism; rickets; cretinism | (1) Clinical<br>(2) Specific investigations | That of specific condition | |
| Solid masses<br>(a) Renal | Hydronephrosis; polycystic kidneys; Wilms' tumour; ectopic kidney | (1) Clinical<br>(2) I.V.P. | That of specific condition | |
| (b) Suprarenal | Neuroblastoma | (1) Clinical<br>(2) I.V.P. | That of specific condition | |
| (c) Spleen | Lipoidoses; gargoylism; acholuric jaundice; Banti's disease; leukaemia; Hodgkin's disease; malaria; kala-azar; glandular fever | (1) Clinical<br>(2) Blood count | That of specific condition | |
| (d) Liver | Cirrhosis; malignant disease; Hodgkin's disease; von Gierke's disease | | That of specific condition | Depends on primary disease |
| (e) Gut | Faeces from constipation; Crohn's disease; appendix disease | | That of specific condition | Depends on primary disease |
| (f) Peritoneal | Retroperitoneal sarcoma; lymphosarcoma; tuberculous glands; psoas abscess; Hodgkin's disease | Clinical | That of specific condition | Depends on primary disease |
| Abdominal wall<br>(a) Hernia | Umbilical and epigastric | | That of specific condition | Depends on primary disease |
| (b) Neoplasms | Lipoma and Hodgkin's disease | | That of specific condition | Depends on primary disease |
| (c) Inflammation | Cellulitis | | That of specific condition | Depends on primary disease |

# ANAEMIA

Children readily become anaemic because of the demands of body growth, disorders of intestinal absorption or inadequate diet.

It must be appreciated that 'anaemia' is only a symptom and that it is essential to determine the cause before any treatment can be given.

## CAUSES

Any classification must be a practical one to help the doctor in the problem of the child with anaemia. The following is based on the mechanisms producing the anaemia.

### DEFECTS OF BLOOD FORMATION

Haemoglobin is a protein containing iron and therefore the body requires adequate supplies of iron in the diet for its manufacture. In addition vitamin $B_{12}$ and folic acid are necessary for the normal maturation of the red cells.

*Iron-deficiency Anaemias*

Here the red blood cells are normoblastic and microcytic. These are by far the most frequent types of anaemia. They may result from nutritional factors such as lack of iron-containing foods. This type of anaemia is frequent in premature infants because of the inadequate store of iron and because of its poor utilization. Inadequate absorption in association with gastro-intestinal upsets is another cause.

*Macrocytic and Megaloblastic Anaemias*

These anaemias result from lack of vitamin $B_{12}$ and folic acid. They are present in steatorrhoeas associated with coeliac disease, in severe liver disease and in kwashiorkor.

*Anaemias due to Marrow Depression*

Marrow depression will also result in anaemia even if there are adequate amounts of raw materials. This is usually one of two types —the aplastic type, where there is no production of blood cells, and the hypoplastic type, where production is seriously reduced.

*Aplastic anaemias*—These may be due to idiopathic disease; drugs, e.g. sulphonamides, chloramphenicol, gold, tridione; irradiation.

*Hypoplastic anaemias*—These anaemias comprise:

(1) Leukaemia and neoplastic conditions such as Hodgkin's disease, which cause mechanical displacement of marrow tissue and interfere with blood formation.

(2) Infections, both acute and chronic, which cause anaemia, probably by interfering with blood formation in the marrow.

### BLOOD LOSS

Loss of blood in acute or chronic haemorrhage causes anaemia. Such situations occur in the haemorrhagic diseases and in bleeding from the gastro-intestinal tract as in hiatus hernia.

### BLOOD DESTRUCTION

Excessive destruction of red blood cells may occur either on account of some intrinsic abnormality of the cells, as in congenital haemolytic anaemias, sickle-cell anaemia and spherocytic anaemias (acholuric jaundice; see chapter on Jaundice), or from some extra-corpuscular mechanism such as haemolysis in infections, blood group incompatibilities and various auto-immunization reactions.

## PATHOLOGY

The functional disturbances of anaemia depend primarily on the condition responsible for the anaemia. The anaemia itself will result in a vague state of ill-health and malaise accompanied by pallor. It is surprising how low the level of haemoglobin can fall before the condition becomes apparent clinically. Anaemias with levels of haemoglobin around 70 per cent are often missed clinically and it is not unusual for a child to be brought for medical attention only when the level of haemoglobin has fallen to 40 per cent.

The normal changes in the levels of haemoglobin at different ages must be appreciated. The haemoglobin level at birth is much higher than in the adult (14·8 g per 100 ml = 100 per cent) and is around 17–20 g. From the second week onwards there is a steady fall in the haemoglobin level until it reaches 11–12 g (77–84 per cent) at 8 weeks. This fall is due to temporary reduction of erythropoiesis rather than to any increase in breakdown of red blood cells. This low level of haemoglobin (11–12 g) is maintained until about 18

months to 2 years of age. From this time the level slowly rises to reach the normal of 14·8 g at puberty.

## DIAGNOSIS

Bearing in mind three significant questions—Is there anaemia present? If so, what is its cause? How should it be managed?—the diagnostic approach may now be considered.

*Is there Anaemia Present?*

An accurate clinical diagnosis of 'anaemia' is difficult and open to gross errors. Pallor is a very common feature in children and is far more often due to causes other than anaemia such, for example, as natural colouring, the emotional state of the child and the state of the lighting in the examination room. It is notoriously difficult to try to estimate the haemoglobin level clinically and no attempts should be made to do so.

If anaemia is suspected because of the appearance or because of symptoms such as general malaise and debility, then its presence or absence must be determined as soon as possible by a haemoglobin estimation and followed up by a more detailed blood examination.

Having confirmed the presence of anaemia by finding a haemoglobin level of below 10 g or 70 per cent a clinical attempt should be made to arrive at a probable cause before undertaking further detailed haematological investigations. Inquiries must be made on the antenatal health of the mother, for maternal anaemia predisposes anaemia in the child. Other predisposing factors are prematurity and twin pregnancies. Haemolytic disease of the newborn may be mild and missed, so a knowledge of the mother's blood groups is important.

The past management of the child must be inquired into and questions asked about diet and supplements. Over-long feeding on milk alone inevitably leads to a severe nutritional anaemia.

The possibility of other diseases such as hiatus hernia, leukaemia, coeliac disease, chronic infections and haemorrhagic disorders must be considered and suitable investigations carried out.

*Examination*

A clinical examination should, in addition to routine procedures, pay special attention as to whether the liver and the spleen are enlarged.

The spleen may be enlarged in leukaemia, Hodgkin's disease,

acholuric jaundice, acquired haemolytic anaemias, chronic liver conditions with portal hypertension, in chronic infections (particularly in subacute bacterial endocarditis and malaria), in thrombocytopenic purpura, and in disorders of metabolism such as amyloid disease and storage disorders which may be associated with anaemia.

Enlargement of the liver is present in most of the conditions just listed with the exception of the acquired haemolytic anaemias and thrombocytopenic purpura.

*Ancillary Investigations*

Ancillary investigations should test for the presence of occult blood in the faeces in cases of gastro-intestinal bleeding and for urobilinogen in the urine in haemolytic anaemias. A barium swallow and meal are necessary to demonstrate whether a hiatus hernia is present.

The more detailed blood examination must include a further haemoglobin estimation, red blood cell count, a total and differential white cell count and examination of the blood film. When haemolytic anaemia is considered a possibility a reticulocyte count and a Coombs' test and determination of red blood cell fragility are necessary.

The platelet count is low in thrombocytopenic purpura and also in aplastic anaemia. The mean corpuscular volume (M.C.V.) determines the average volume and shows most accurately whether the anaemia is macrocytic, normocytic or microcytic. The mean corpuscular haemoglobin concentration (M.C.H.C.) measures the percentage of haemoglobin in the red blood cell and is the truest indicator of whether iron deficiency is present or not.

For the diagnosis of the great majority of anaemias we require a haemoglobin level, M.C.V. and M.C.H.C., a white blood cell count and a stained blood film. An examination of the bone marrow, however, will be required to confirm a number of types such as aplastic anaemia and leukaemia.

## SIGNIFICANCE

Anaemia is a very frequent condition in children. In the majority of cases the condition is a simple iron-deficiency anaemia due to nutritional factors. It is important that the condition should be diagnosed as early as possible, for the response to treatment is excellent.

The possibility of anaemia should always be kept in mind and

suitable facilities should be available and used to confirm its presence.

Although iron-deficiency nutritional anaemia is the most frequent type a more complete blood examination is required if other types are not to be missed.

## SPECIFIC TYPES

### IRON-DEFICIENCY ANAEMIA

Iron-deficiency anaemia is by far the most common type. Its exact frequency is unknown because no large-scale studies have been carried out, but it is estimated to be present in 10–15 per cent of all infants.

It is more frequent in boys than in girls and the maximal incidence is in infancy, between 3 months and 4 years of age.

*Aetiology*

Because of rapid growth the baby's iron balance is very precarious for the first two years and is easily upset.

*Prenatal factors*—Premature infants have a relatively low store of iron at birth and unless this is made good by oral iron anaemia is very likely to occur. The same situation occurs in multiple pregnancies where the available iron has to be shared.

*Postnatal factors*—Milk contains little iron and if mixed feeding is delayed beyond the fourth month anaemia is very likely to occur. Absorption of iron may be interfered with in recurrent infections. Chronic blood loss is a rare cause.

*Blood Picture*

The blood picture is of a hypochromic anaemia with a M.C.H.C. below the normal of 32–38 per cent, usually with microcytosis and the presence of nucleated red cells in the peripheral blood.

*Clinical Features*

The characteristic appearance is that of a pale but well nourished, often fat, baby. The mother complains of the baby's poor appetite and general lassitude and the liability to recurrent minor infections.

Physical examination usually reveals nothing very much apart from pallor. Blowing systolic heart murmurs may be heard all over the precordium whenever the haemoglobin level is below 50 per cent.

*Course*

Without treatment the baby will have a stormy infancy with recurrent infections which will themselves aggravate the anaemia. Natural improvement will occur late in childhood from about 6 years onwards.

With treatment there is a gratifying improvement in the appearance. Colour returns to the cheeks and the baby looks generally better. Appetite also improves and there is much less liability to the common infections.

## LEUKAEMIA

An early feature of leukaemia is anaemia. It is relatively uncommon and on average the general practitioner may expect to meet a new case every 7–10 years. The incidence is increasing, however, and any sick child with anaemia, haemorrhages and enlargement of the spleen should be investigated so that leukaemia can be excluded.

*Aetiology*

The aetiology is unknown but there are some suggestions that it may be associated with some abnormality of the chromosomes, thus it is said to be more frequent in mongols and it may be related to excessive irradiation from natural and therapeutic sources.

*Age*

Leukaemia may occur at any age but in children it is most common between 3 and 5 years of age, becoming increasingly less frequent until adolescence.

*Clinical Features*

Although attempts are made to subdivide leukaemia into acute and chronic varieties, and further to classify the latter type according to the predominant cells, in children acute leukaemia is by far the most usual variety with immature lymphatic cells.

*Onset*—the presenting features are lassitude, malaise and pallor. Pains in the legs and chest are also frequent symptoms. Nose-bleed and petechiae may occur.

*Examination*—On examination the child is pale, and obviously anaemic. The progress of the anaemia may be very rapid, the haemoglobin level dropping 30–40 per cent over a week. Weight is not lost and the child appears well covered. Bruising or petechiae may be present. The spleen is enlarged and firm in consistency. Lymph glands in the neck and axillae and groins are enlarged. The

mouth and throat tend to become infected in the later stages. This infection is subacute with little evidence of any acute reaction. The throat tends to be covered by a dirty grey offensive slough without much redness and swelling of the tissues.

*Blood Picture*

The diagnosis is confirmed by examination of the blood. There is usually a profound anaemia, the haemoglobin level is often below 30 per cent. This is normocytic and normochromic. The white cell count, contrary to expectations, is often not raised above 20,000–50,000 but may be lower and, of course, much higher on occasions. The characteristic feature is the presence of abnormal primitive white cells in the peripheral blood and the bone marrow.

*Prognosis*

The prognosis is hopeless. Death will occur within weeks without treatment and within the year in most cases even with treatment.

*Differential Diagnosis*

Differential diagnosis must be made from glandular fever, where there are many characteristic mononucleated white cells in the blood and the Paul–Bunnell test is positive. In whooping cough there may be a lymphocytosis of up to 100,000 per ml but these cells are mature and there are other clinical features such as cough, whoop and vomit. The various types of purpura, thrombocytopenic and anaphylactoid, and agranulocytosis may present similar features, the diagnosis being made by examination of the bone marrow.

## APLASTIC ANAEMIA

In aplastic anaemia there is failure of red cell production in the bone marrow. The red marrow is replaced by a yellow gelatinous substance and marrow puncture and smears show absence of erythropoietic tissues. The peripheral blood reveals a progressive anaemia, normocytic and normochromic in type with no reticulocytes or nucleated red cells. Associated with this anaemia there are agranulocytosis and a low platelet count.

*Aetiology*

The chief causes of aplastic anaemia are:

(1) Chemical poisons, e.g. sulphonamides, chloramphenicol, tridione.

(2) Over-irradiation with x-rays, radium or other radio-active materials.

(3) Severe infections, which very rarely terminate in an aplastic anaemia.

(4) Marrow displacement by neoplastic tissues or osteopetrosis.

*Clinical Features*

The clinical features are those of a severe anaemia, with extreme pallor. Agranulocytosis with necrotic ulceration of the throat and skin may occur. Thrombocytopenic purpura with haemorrhages may also be a feature.

*Diagnosis*

This depends on the blood picture, the bone marrow smear, a history of possible contact with causal agents and lack of evidence of haemolysis with no urobilinogen in the urine and a negative Coombs' test.

## HAEMOLYTIC ANAEMIAS

The red blood cell has a life of about 120 days and at the end of that time it disintegrates and the fragments are absorbed by cells of the reticulo-endothelial system, i.e. spleen, liver and bone marrow.

The liberated haemoglobin is broken up into two parts—iron which is retained and bilirubin which is excreted by the liver into the biliary system and then into the gut where it is reduced to urobilinogen. Some of this is reabsorbed and excreted in the urine and the remainder in the faeces.

In haemolytic states where an excessive number of red cells are broken down the amount of bilirubin in the blood is abnormally high as is the level of urinary urobilinogen.

If haemolysis is very rapid and very large numbers of cells are destroyed there is no time for the haemoglobin to be broken down and it is excreted as such in the urine, which becomes black (haemoglobinuria). In chronic haemolysis, where the process of destruction of red cells is slower, the accumulation of bilirubin and urobilinogen occurs as described. The bone marrow attempts to produce extra red cells in response to the anaemia and a reticulocytosis occurs (above the normal limit of 2 per cent). To cope with the destroyed cells the spleen enlarges and becomes palpable.

*Diagnosis*

The diagnostic features of a haemolytic anaemia, then, are an

anaemia with reticulocytosis, jaundice with a high serum bilirubin and high urobilinogen in the urine and no bile pigments and splenomegaly. The faeces are normal in colour but with an excess of stercobilin. The usual types of haemolytic anaemias are:

(1) Congenital haemolytic disease of the newborn.
(2) Acholuric jaundice (congenital haemolytic anaemia).
(3) Acquired haemolytic anaemias.
(4) Sickle-cell anaemia and thalassaemia (or Cooley's anaemia).

### ACHOLURIC JAUNDICE

Acholuric jaundice (congenital haemolytic anaemia) is a rare familial disease in which the essential abnormality is in the fragility of the red blood cells. It is transmitted as a Mendelian dominant in both sexes but not all progeny are affected. The red blood cells are spherical (spherocytosis) and fragile and are readily destroyed in the spleen, which is enlarged.

*Clinical Features*

Although it has been described in infancy, as a rule the condition does not become obvious until later, from the age of 5 years onwards.

The case may present in one of the following ways—mild jaundice or anaemia, an enlarged spleen or during an 'aplastic crisis'.

During the 'latent periods' the child is somewhat poorly developed with a sallow skin and icteric conjunctivae. A firm spleen is usually palpable and sometimes in older children chronic indolent ulcers appear on the shins. With the persistently high level of bilirubin in the blood there is a tendency to form pigment stones in the gall-bladder and biliary colic may be a feature.

At times, triggered off by infections, cold or fatigue, 'aplastic crises' occur with malaise, fever, abdominal pain, increase in jaundice and anaemia. They tend to last for a few weeks and then the condition settles down to the latent stage.

*Blood Picture*

The blood picture reveals a low haemoglobin, around 60 per cent. The red cells are small in diameter but thick and convex. Reticulocytosis is a feature and is usually above 10 per cent. A fragility test shows that the red blood cells are abnormally susceptible to haemolysis by hypotonic saline solution—haemolysis commencing at 0·75 per cent concentration, as opposed to 0·45 per cent in the

normal, and being complete at 0·4 per cent compared with 0·3 per cent in the normal. Serum bilirubin is increased. The urine contains no bile but urobilinogen is increased. The Coombs' test for the presence of haemolysin antibodies is negative.

*Diagnosis*

The diagnosis should be considered in an anaemic child with jaundice and a large spleen. A history of biliary colic in a child should make one think of acholuric jaundice. A positive family history is helpful but a negative one does not exclude the diagnosis.

## ACQUIRED HAEMOLYTIC ANAEMIAS

*Aetiology*

This group of anaemias is caused by the presence of haemolysing agents in the blood, the red cells themselves being normal. Two types of cause can be distinguished:

(1) Exogenous causes, such as malaria, septicaemia and such chemicals as sulphonamides, quinine, phenylhydrazine, lead, naphthalene (in moth balls) and potassium chlorate.

(2) Endogenous haemolysins, which are specific antibodies produced within the body and which have the ability to attach themselves to, and to destroy, the red cells. Well known haemolysins are those found as a result of mismatched blood transfusions. Often, however, the provoking cause is unknown but the presence of these antibodies is shown by a positive Coombs' test.

*Clinical Features*

Acute haemolytic crises present with sudden malaise, fever, pains in back and limbs, pallor, vomiting and diarrhoea; leucocytosis may be a feature. This type of haemolytic anaemia is known as Lederer's anaemia. It is treated by blood transfusions.

Chronic haemolytic anaemias present similar clinical features to acholuric jaundice, and may be controlled by corticosteroids.

## SICKLE-CELL ANAEMIA

This condition occurs mainly in Negroes. The red cells sickle when the oxygen tension is lowered. The haemolytic anaemia is often very severe. Large ulcers on the ankles are frequently seen and joint pains are also common. The spleen is enlarged in infants. Haemolytic crises can occur and the sickling of the cells may cause obstruction of small blood vessels in the brain, kidneys, lungs and liver.

### THALASSAEMIA

Thalassaemia (or Cooley's anaemia) is found among those of Mediterranean stock. The anaemia is microcytic and hypochromic in type and there is evidence of haemolysis. The liver and spleen are enlarged in severe cases and in these patients the prognosis is poor. In others the trait may be latent and the prognosis is better.

### TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Defects of blood formation* | | | | |
| Iron-deficiency anaemia | Frequent male predomination; maximal in infancy and up to 5 years of age; pallor and poor general health are main features | (1) Suspect it clinically (2) Confirm by blood examination | Oral iron | Excellent response to iron therapy |
| Macrocytic anaemia | Rare—May occur in malabsorption disorders, such as coeliac disease or severe liver disease | (1) Blood (2) Marrow | (1) Treat primary disease (2) Vitamin $B_{12}$ or folic acid | Depends on primary cause |
| Aplastic anaemia | Rare—Result of depression of bone marrow activity from drug poisoning or in association with leukaemia or severe infections | (1) Blood (2) Marrow | (1) Treat primary cause (2) Blood transfusion | Bad outlook |
| *Anaemias from blood loss* | Follow acute or chronic bleeding; occur in haemorrhagic diseases and hiatus hernia or following trauma | (1) Blood (2) Occult blood in stools | Treat the cause | Depends on the cause |
| *Haemolytic anaemias* | | | | |
| Acholuric jaundice | Rare—Congenital fragility of red blood cells which are spherocytic; clinically these are mild anaemia, jaundice, enlarged spleen, gall-stones, ulcers of legs | Blood: spherocytosis reticulocytosis, fragility | Splenectomy may be of value | Doubtful course; chronic ill-health |
| Drugs and poisons | Rare—May occur from sensitivity reactions or direct poisoning, e.g. sulphonamides, lead, potassium chlorate | (1) Blood (2) History | Blood transfusion | Doubtful outlook |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Sickle-cell anaemia | Rare—Congenital trait; mainly in Negroes; may be very severe; spleen enlarged in some; haemolytic crises may occur | Blood: sickle cells | No specific treatment | Doubtful outlook; chronic ill-health; haemolytic crisis |
| Thalassaemia | Rare—Congenital trait in those of Mediterranean stock; severity varies, may be latent or severe with death | Blood | No specific treatment | Doubtful outlook; chronic ill-health; haemolytic crisis |
| Idiopathic acquired haemolytic anaemia | Rare—Cause unknown; may be acute or chronic | Blood | (1) Blood transfusion (2)Steroids (3) Splenectomy considered in chronic cases | Doubtful outlook |

# BACKWARD CHILDREN

Mental retardation is often more an educational and a social problem than strictly a medical one. It is up to the physician to make the early diagnosis and to decide on the possibilities of specific treatment. The management of the child as a whole, however, must always be a joint effort with social and other workers.

This problem is more important now than ever before, because as more research is being carried out so specific conditions are being defined and preventive and therapeutic measures discovered with hopes of eventual normal development.

## CAUSES

There are a very large number of conditions and groups of conditions in which mental retardation is a feature. In some it is the main clinical feature and in others it is one part of a syndrome. The following list is by no means a comprehensive one and is liable to be altered as a result of research that is taking place in this field.

*Physiological*—Physical and mental development in children do not follow any rigid or regular schemes. Marked variations may occur in the times at which certain milestones of development are passed. No child should ever be regarded as mentally retarded if he is backward in only one or two fields of development providing that he is normal in other fields. The mentally retarded child is backward in *all* fields of development, though this backwardness is more evident in some, such as speech, than in others, particularly locomotion.

*No known cause* (*simple amentia*)—This group comprises about 80 per cent of all mentally retarded children. The severity varies from 'slight backwardness' to the child who is quite unable to do anything for himself. This large group probably contains many subgroups which will be separated when their aetiology is defined.

*Chromosomal anomalies*—Mongolism has been shown to be associated with an extra chromosome (associated with chromosome No. 21, hence a new suggested name for mongolism is '21-trisomy'). It is probable that other syndromes with mental retardation will be shown to be due to a similar cause.

*Metabolic disorders*—This is another group of causes of mental

retardation that has recently been defined and many specific subgroups are being separated. It must be appreciated that all these abnormal states are very rare and account (as yet) for a very small proportion of the total. Examples are phenylketonuria, aminoaciduria, maple syrup urine disease, galactosaemia, gargoylism, sucrosuria, hepato-lenticular degeneration, idiopathic hypercalcaemia.

*Infections*—Meningitis and encephalitis may lead to mental retardation. A special type is the encephalitis seen after specific fevers and following immunization against whooping cough. (See chapter on Headache.)

*Congenital abnormalities*—Abnormalities such as hydrocephalus may be associated with mental retardation, but this is by no means always so. Other structural defects may include craniostenosis, hypertelorism and microcephaly.

*Brain damage*—Birth trauma may damage the brain and result in mental retardation. Other conditions associated with damage to the brain are subdural haematoma, which may cause mental retardation if untreated, and cerebrovascular thrombosis.

*Antenatal infections*—Infections such as rubella, toxoplasmosis and syphilis are potential causes of mental retardation.

*Endocrine abnormalities*—Cretinism, gonadal dysgenesis, hypoparathyroidism, hyperparathyroidism, hypoglycaemia and such endocrine abnormalities are often associated with mental retardation.

*Epilepsy*—Mental retardation is rare in idiopathic epilepsy. It may occur in cases where the epilepsy is secondary to some other disease which causes retardation. (See chapter on Convulsions.)

*Cerebral palsy*—Only about half of these children are mentally retarded.

*Miscellaneous conditions*—Many other conditions may cause mental retardation; they include eye lesions such as retrolental fibroplasia, neuromuscular disorders, Fanconi's anaemia, achondroplasia, arachnodactyly, congenital heart disease and lead poisoning.

## PATHOLOGY

The pathology of mental retardation is related to the causal condition.

## DIAGNOSIS

An early and correct diagnosis of mental retardation is never easy and should rarely be made in a hurry on the basis of a single examination. Even features of apparently obvious mongolism may at

times be erroneously interpreted. It is much better to follow the child for a while to observe progress before giving the diagnosis and prognosis to the parents.

The whole situation must be considered. The diagnosis must never be made on the basis of slowness or retardation in one or two fields. An accurate diagnosis of development is always a personal and individual technique. Contact and good personal relations must be built up with the mother and child. A thorough knowledge and appreciation of the different rates of progress that are possible are necessary and a routine should be followed.

*History and Examination*

A history should aim at picking out approximate times at which recognizable milestones were passed, such as smiling, taking notice, lifting the head, grasping, sucking, self-feeding, speaking, walking and dressing.

The physical appearance should be studied to discover possible leading signs such as mongolism, hydrocephalus, microcephalus and cretinism. Tests for metabolic disturbances should be carried out wherever possible.

In older children intelligence, educational performance and general knowledge tests will assist in the assessment.

### 'PSEUDORETARDATION'

Differentiation of mental retardation must be made from certain sensory and allied defects which may erroneously suggest the presence of mental defects.

*Sensory defects*—Blind infants at first take little interest in their surroundings and may be thought to be mentally retarded. Diagnosis of deafness is extremely difficult in infancy and confusion may occur because mentally normal deaf children may be retarded because they cannot use hearing in their development.

*Language disabilities*—Slow development of reading and spelling is often quite unrelated to any general mental retardation. Aphasia may be caused by local lesions in the brain.

*Cerebral palsy*—It is quite wrong to assume that all these children are mentally retarded. Severe retardation only exists in about one half of them.

## SIGNIFICANCE

The significance of the discovery of backwardness is grave and the diagnosis must never be made too readily but only after careful consideration. The diagnosis will be a serious matter for the child and

parents and if it proves to be incorrect in the future they will never forgive the mistake.

In all situations efforts must be made to discover causes which may be treatable such as some of the metabolic disorders where attention to the diet and avoidance of certain offending foods is essential if permanent mental damage is to be avoided.

*Prolonged illness*—Prolonged illness, malnutrition and social deprivation may all lead to significant symptomatic developmental retardation especially in infancy. If the causes can be corrected the child soon catches up and develops normally.

*Psychological factors*—Emotional factors are not infrequently responsible for a disparity between a child's actual performance and his inate ability. Environmental deprivation in infancy is particularly likely to cause secondary developmental retardation.

Psychotic illness such as schizophrenia is an important cause of 'pseudo-feeblemindedness'.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Physiological | Slowness in development in one or two fields is normal | Clinical and that of primary disease | Reassurance | Normal development |
| No known cause | Accounts for 80 per cent of mentally retarded children; severity varies | Clinical and that of primary disease | General care | Poor outlook |
| Mongolism (chromosome defects) | Due to abnormalities in chromosome No. 21; specific clinical condition | Clinical and that of primary disease | General care | Poor outlook |
| Metabolic disorder | Rare—Important group; if specific type can be defined then prevention of mental retardation may be possible | Clinical and that of primary disease | Specific measures possible | Prevention of mental retardation possible |
| Infections | Following meningitis or encephalitis | Clinical and that of primary disease | General care | Poor outlook |
| Brain damage | Associated with birth trauma, hydrocephalus, cerebrovascular causes | Clinical and that of primary disease | General care | Poor outlook |
| Antenatal infections | Maternal rubella, toxoplasmosis and syphilis | Clinical and that of primary disease | General care | Poor outlook |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Endocrine abnormalities | Cretinism, gonadal dysgenesis, disorders of parathyroids and hypoglycaemia | Clinical and that of primary disease | Specific measures possible | Poor outlook in spite of possibilities of specific therapy |
| Epilepsy | Mental retardation in severe epileptics, especially where it is secondary to some primary disease | Clinical and that of primary disease | Control of epilepsy | Doubtful prognosis |
| Cerebral palsy | Only about half are mentally retarded | Clinical and that of primary disease | General care | Prognosis more hopeful than in the past |
| Miscellaneous | In association with retrolental fibroplasia, neuromuscular disorders, Fanconi's anaemia, achondroplasia, arachnodactyly, congenital heart disease and lead poisoning | Clinical and that of primary disease | Depends on primary condition | |

# COMA

Alterations in consciousness in infants and children include delirium, stupor and coma.

Delirium is a state of confusion, disorientation and general excitability. It accompanies many acute illnesses associated with fever and toxicity.

Stupor is a state of unconsciousness from which the child may be momentarily aroused.

Coma is a state of profound and prolonged unconsciousness, often preceded by a period of stupor. It presents a rare but important clinical emergency demanding urgent diagnosis and careful management.

## CAUSES

*Trauma*

Children are frequently involved in accidents on the road, at play and in the home. The head is often receiving blows, most of which have no harmful effects. It is only rarely that damage to the brain occurs.

*Cerebral concussion, contusion or laceration*—These conditions are usually followed by immediate loss of consciousness and coma.

*Subdural haematoma*—This condition is a likely cause of persistent symptoms and signs after a head injury. Headache, vomiting, unilateral pupillary dilatation and other localizing signs may be present. Loss of consciousness may be delayed for a few days or even weeks after the injury. When suspected, urgent surgical drainage of the clot is required.

*Extradural haematoma*—This leads to swelling of the scalp, hemiparesis, progressive loss of consciousness, unequal pupils, with dilatation on the involved side, and finally convulsions and death. First signs appear within minutes or hours after the injury, but there may be a lucid interval.

*Infections*

*Toxic encephalopathy*—A toxic encephalopathy may occur in the course of severe infections with septicaemia.

*Intracranial abscess*—An intracranial abscess may develop by local

spread from an ear infection or from a distant focus of infection elsewhere in the body. Children with congenital cyanotic heart disease are especially prone to this complication.

*Meningitis*—Meningitis may present with classical signs of headache, vomiting, loss of consciousness and neck stiffness, but in infants the illness may be very vague with few local signs. (See chapter on Headache.)

*Encephalitis*—This may occur in association with the common fevers, especially mumps, or follow immunization against whooping cough and smallpox or some other virus infections. Clinically there is evidence both of general involvement (i.e. coma and convulsions) and local involvement (i.e. paralysis of cranial nerves and hemiplegia) of the central nervous system. (See chapter on Headache.)

*Poisons*

Accidental poisoning by drugs such as barbiturates, iron salts and aspirin, or by lead or coal gas may all cause coma. Poisoning should be considered as a possibility in any unexplained coma.

*Intoxications and Physical Agents*

Intoxications and physical agents may also be responsible for coma—such as uraemia, diabetic ketosis and hypoglycaemia, electric shock, heatstroke and exposure to cold.

*Epilepsy and Convulsions*

The period of unconsciousness following convulsions may be prolonged especially in status epilepticus.

*Cerebrovascular Catastrophes*

*Cerebral haemorrhage*—Cerebral haemorrhage may occur either intracranially in the newborn; in the subarachnoid space from a rupture of an aneurysm in the circle of Willis; in the subdural or extradural spaces either from trauma or with haemorrhagic diseases.

*Cerebral thrombosis*—This condition is rare in childhood except in those with polycythaemia due to cyanotic heart disease, or in infants who are severely dehydrated or who have an infection spreading from the middle ear.

*Cerebral embolism*—This is also rare but may occur in bacterial endocarditis or as a result of fat embolism after injury.

*Acute infantile hemiplegia*—This is a rare condition of infants. The onset is sudden with a convulsion followed by coma and eventual

hemiparesis. It is believed to be due to a sudden cerebrovascular occlusion.

*Intracranial Tumours*

Intracranial tumours will cause coma in their late stages but may do so earlier and suddenly because of haemorrhage into the growth.

## PATHOLOGY

The pathological changes in the brain in coma obviously depend on the causal condition. However, in a good proportion of children who die in coma there are no apparent morbid anatomical changes. This absence of abnormal findings occurs in states such as concussion, epilepsy, poisoning and the various intoxications. In these the abnormalities must be intracellular and not amenable to detection by present methods.

The more gross lesions causing coma include traumatic contusion and laceration of the brain, meningeal or intracerebral abnormalities following infection, destructive and degenerative changes from cerebrovascular lesions such as haemorrhage, thrombosis and embolism.

## DIAGNOSIS

In dealing with a child in coma a simple yet systematic plan of approach is the best rather than an attempt at a more complete all-inclusive but impractical scheme.

Possible likely causes should be considered even before the child is examined. This mental exercise serves as a stimulus and avoids unnecessary rush and panic.

*History*

A history must be obtained from the mother. Antecedent illnesses and events will be considered. For example, fulminating and severe infections are suggested by a story of fever, convulsions and some localizing symptoms. Local disease in the central nervous system is suggested by features of meningeal irritation and rising intracranial pressure, i.e. vomiting, convulsions, headache and ataxia. Questions must be asked as to the drugs kept in the house and more precisely as to what medicines and tablets the mother is taking.

Preceding symptoms of diabetes mellitus, renal failure, tuberculosis and malignant disease are all vague and their more accurate assessment must await the examination.

*Examination*

Physical examination should be quick but thorough and serve in the first instance to try to pick out any readily remediable causes such as infections, diabetes mellitus, poisoning and the effects of trauma. The head should be examined for injuries and the tension in the fontanelle estimated for a rise in pressure. The systems must be examined in order. Abnormal signs in the chest may be either primary and causal or secondary and the effects of the coma (or both). Examination of the nervous system must include careful inspection of the optic fundi, for papilloedema, choroidal tubercles and haemorrhages and exudates; pupillary changes may be significant. Tone and reflexes are all reduced in coma and of little help.

The urine must be examined for sugar, ketone bodies and albumin; catheterization may be necessary. Other investigations include examination of the cerebrospinal fluid, but care must be taken in patients who are shocked and who have papilloedema, because in these lumbar puncture may cause serious complications. A radiographic examination of the skull is indicated whenever there is a history or suspicion of head injury. If the possibility of poisoning exists, gastric lavage may be necessary for diagnostic as well as therapeutic reasons. An electroencephalogram may be necessary in patients with obscure or unexplained coma.

## SIGNIFICANCE

Prolonged unconsciousness in coma is always of the greatest significance and the child must be urgently admitted to hospital for investigation on the lines stated.

## SPECIFIC CONDITIONS

### DIABETIC COMA

Although no child, or adult, should ever die from diabetic coma, it still accounts for an appreciable mortality in diabetes mellitus. To avoid this, early diagnosis and management of the diabetes is essential.

The clinical features of diabetes mellitus in children are the rapid onset, the symptoms becoming increasingly severe over one or two weeks; polyuria and thirst are the main symptoms together with general malaise and loss of weight and pruritus vulvae in girls. All these symptoms merit testing the urine for sugar.

Diabetic coma may occur as the presenting feature in an unknown

diabetic, but more often it occurs as a complication of diabetes in association with infections or with errors of management in cutting down or stopping insulin for the wrong reasons, i.e. during times when the child is not eating.

*Clinical Features*

The child feels ill with headache. Nausea and vomiting are prominent features in the pre-coma stage. Abdominal pain may be severe and diabetes must be considered in the differential diagnosis of an 'acute abdomen'. Drowsiness and restlessness follow and then coma which deepens every hour.

*Biochemical Disturbances*

Biochemical disturbances follow the hyperglycaemic ketosis together with the vomiting and polyuria. These comprise:

(1) Dehydration and haemoconcentration as evidenced by a raised haemoglobin concentration.

(2) Depletion of fixed base and chloride, leading to acidosis and lowering of the carbon dioxide combining power of the plasma.

(3) Very high blood sugar—well over 750 mg per 100 ml.

(4) Large amounts of sugar and acetone in urine.

(5) Fall in blood potassium.

*Treatment*

Treatment is very urgent and requires insulin to control the diabetes, electrolytes and fluid to correct the biochemical upsets, and dehydration and treatment of the precipitating cause.

## TUBERCULOUS MENINGITIS

Several factors have combined to reduce the incidence of tuberculous meningitis, including improved social conditions, more intensive public health measures, prophylactic and specific treatment of primary tuberculous infections and inoculation with B.C.G. vaccine.

Nevertheless, the early diagnosis of such cases is of great importance if permanent disabilities and death are to be prevented, and although coma is a late stage of this condition, it is useful to consider it here.

*Clinical Features*

Three stages of the condition are recognized: early, medium and advanced.

*Early stage*—The common presenting features in the early stages are fever, vomiting, constipation and apathy. These are common disturbances in childhood but tuberculous meningitis should be considered if no adequate explanation is forthcoming. Constipation is invariable and may be severe. Signs of meningeal irritation at this stage are infrequent and the young child (tuberculous meningitis is most common between 1 and 4 years of age) rarely complains of headache. Convulsions may be a feature of the early stage. Diagnosis depends on aroused suspicions. Only if the condition is always in the mind of the doctor when faced with vague and persisting symptoms will diagnostic steps be put in motion. A history of contact is of importance but is not always available. A positive tuberculin skin test is of the greatest significance. A negative result does not invalidate the diagnosis. Choroidal tubercles may be seen in the optic fundi. A chest x-ray may show a primary complex or miliary appearances.

*Middle stage*—In the middle stage neurological signs appear. Meningeal irritation is evidenced by the neck stiffness and a positive Kernig's sign. Cranial nerve palsies, spasticity, photophobia, drowsiness and irritability must be regarded urgently, for delay will lead to various permanent disabilities such as hydrocephalus, blindness, deafness, spastic palsies and mental deficiency.

*Advanced stage*—By the time the child is in coma the outlook is very grave. Confirmation of the diagnosis is obtained by examination of the cerebrospinal fluid. This is clear or slightly opalescent. The cell count is raised (50–500 per $mm^3$) and lymphocytes are the predominating cells. The sugar content is low (below 50 mg per 100 ml). The protein content is high (over 50 mg per 100 ml). Acid-fast bacilli may be seen on a smear or from the coagulant pellicle that forms on standing.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Trauma | Concussion: immediate loss of consciousness<br>Subdural haematoma: persistent symptoms after injury; coma may be delayed for weeks or months<br>Extradural haematoma: short delay after injury and then progressive coma | (1) Clinical<br>(2) Radiograph of skull | (1) Hospital management<br>(2) Possible surgery | Considerable mortality rates |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Infections | Toxic encephalopathy: with severe infections<br>Intracranial abscess: follows primary infection elsewhere, i.e. chest or ear<br>Meningitis: headache, vomiting, neck stiffness, coma or few signs and coma<br>Encephalitis: often a virus infection; convulsions, local C.N.S. signs and coma | (1) Clinical<br>(2) C.S.F. examination | Depends on type of infection | Doubtful outcome |
| Poisons | Accidental poisoning with barbiturates, iron preparations, lead or coal gas | Always consider the possibility of poisoning in coma of uncertain cause | (1) Hospital management<br>(2) Depends on cause | Good outcome if treated early |
| Intoxications and physical agents | Diabetes mellitus, uraemia, hypoglycaemia, electric shock, heat stroke and exposure to cold | (1) Test urine in all cases of coma<br>(2) That of primary condition | Depends on cause | |
| Epilepsy | Period of unconsciousness following epileptic attacks may be prolonged in status epilepticus | Clinical | Non-specific | Poor outlook in recurrent severe attacks |
| Cerebro-vascular catastrophes | Cerebral haemorrhage in newborn into subarachnoid space in congenital aneurysms, subdural or extradural haematomas<br>Cerebral thrombosis associated with congenital heart disease and infections of ear<br>Cerebral embolism in infections or trauma | (1) Clinical<br>(2) C.S.F. examination<br>(3) Special neurosurgical procedures | That of primary cause | Poor outlook |
| Intracranial tumours | Coma either in late stages or as sudden complication of haemorrhage into the tumour | (1) Clinical<br>(2) Lumbar puncture with care<br>(3) Special neuro-surgical procedures | Possible surgery | Hopeless prognosis as most tumours are malignant |

# CONSTIPATION

It is often difficult to decide when children are constipated. One has to rely on the mother's history. Some mothers will complain that their infants are constipated when normal stools are passed at what they consider to be infrequent intervals.

Constipation may be considered to be present when hard stools are passed and when they lead to straining and pain on defaecation. Constipation is also present when defaecation is incomplete rather than infrequent. Sometimes there is a form of faecal impaction. A hard mass is left behind in the rectum and only small amounts of fluid faeces are passed. Constipation is also used to describe the complete absence of defaecation that occurs in intestinal obstruction.

## CAUSES

### NEWBORN

*Physiological*—Although normally some meconium is passed within a few hours of birth, occasionally this may be delayed for 24 hours or longer. If this occurs the passage of a well-greased little finger will release a mass of meconium. It has been postulated that this delay is caused by a 'tight' sphincter but it is doubtful whether any abnormality is actually present.

*Rare* causes are as follows:

(1) Imperforate anus. External appearances may not be abnormal when the obstruction is high in the rectum.

(2) Atresia of gut or intestinal obstruction. No meconium is passed. Vomiting occurs and this is early in high obstruction and within 48 hours in a lower intestinal block.

(3) Hirschsprung's disease. This may present as failure to pass meconium with abdominal distension.

### INFANCY

*Physiological*—It is quite normal for healthy breast-fed babies to have their bowels open only once every 2–7 days.

*Underfeeding*—Underfeeding with insufficient fluid and bulk is a frequent cause.

*Anal fissure*—Anal fissure causing pain on defaecation will lead to constipation.

*Rare* causes are congenital pyloric stenosis; mental deficiency (including cretinism); Hirschsprung's disease.

### CHILDHOOD

*Physiological*—From dietetic causes, and faulty training and upset in routine.

*Emotional causes*—The 'potting couple'.

*Symptomatic*—In fevers and other illnesses with reduced intake of food and fluids.

*Rare* causes are idiopathic megacolon; mental deficiency; Hirschsprung's disease; chronic intestinal obstruction due to organic causes; neurological diseases which affect reflex mechanisms and the anal sphincter, leading inevitably to constipation—this occurs in meningocele, spina bifida occulta, cerebral palsy, poliomyelitis and various myopathies.

## PATHOLOGY

The disturbances resulting from constipation are both real and imaginary. Many of the imaginary disturbances are based on misconceptions and prejudices. The complaints coming from the mother are usually based on her own anxieties and fears.

The idea that children need to be 'trained' dies hard. Mothers still believe that strict training is necessary for bowel control. This tends to lead to friction between mother and child and rebellion on the part of the latter with many and various symptoms, the chief of which is refusal to pass motions. The other symptoms are erroneously attributed to constipation. This symptom of 'constipation' is then treated by laxatives on the mistaken and exaggerated idea of the importance of the regular daily motion. Motions are carefully examined by the mother who then makes the difficult assessment as to whether the amount passed has been sufficient. All this ritual causes an aggravation in the vicious cycle with constipation, anxiety in the mother, rebellion and refusal to pot in the child, leading to more constipation.

The functional disturbances of mild constipation due to such minor causes are relatively insignificant and include pain on defaecation and vague abdominal discomfort.

In serious disorders causing constipation, such as Hirschsprung's disease, the effects are those of a chronic intestinal obstruction,

with distension, electrolyte disturbances, failure to thrive and anaemia.

## DIAGNOSIS AND SIGNIFICANCE

The diagnosis of the cause and the significance of constipation depend on a careful clinical assessment.

*History*

In the history the duration of the condition is of importance. A sudden onset of constipation in a previously healthy child should never be dismissed lightly, especially if accompanied by abdominal pain and vomiting. This combination is very suggestive of an intestinal obstruction. Constipation with abdominal distension in boys dating from birth should make the diagnosis of Hirschsprung's disease a possibility.

Chronic constipation is an insignificant symptom in a child who is generally well and obviously in good health in spite of a multitude of vague symptoms that the mother may list. Where the child is unwell a primary cause should be sought.

Vomiting in association with constipation makes the situation much more serious, for it suggests the possibility of some intestinal obstruction or some more generalized disorder.

*Examination*

On examination the presence of abdominal distension should be noted, for this suggests chronic obstruction. Faecal masses may be felt and must be distinguished from other abdominal tumours. Rectal examination is essential. It may reveal an empty rectum in Hirschsprung's disease or more usually one filled with hard faeces as in idiopathic megacolon and the functional type of constipation. An anal fissure causing painful defaecation can only be seen by careful inspection; there is spasm on rectal examination.

## SPECIFIC CONDITIONS

### CONGENITAL ABNORMALITIES OF THE ANUS AND RECTUM

Severe constipation with dilatation of the colon may result from certain congenital malformations. These may be of different degrees: (1) Imperforate anus, where the rectum and anus are normally formed but separated by a membrane, or very rarely there may be complete absence of the anal canal. (2) Rectal agenesis, where there

has been no development of the rectum from the level of the levator ani muscles downwards. (3) Rectal agenesis with a fistula, in which the rectum also fails to develop but a fistulous communication exists between the bladder or vagina and the blind end of the pelvic colon. (4) Ectopic anus, which is associated with an imperforate anus. The anal opening may be situated anywhere along the median perineal line.

In all these situations symptoms date from birth, but complete constipation is absent in incomplete malformations where stenosis is present rather than agenesis.

Constipation is complete, except in rare instances where the obstruction is partial. Progressive abdominal distension soon occurs, but vomiting may be delayed up to 48 hours. Where rectovesical fistula is present meconium-stained urine is a characteristic feature.

Early recognition may be easy by inspecting the perineum and attempting a rectal examination, but with a lesion higher up the external appearances may seem normal. Determination of the exact anatomical derangements is more difficult, requiring careful radiograph studies before any surgical treatment is undertaken.

### HIRSCHSPRUNG'S DISEASE (CONGENITAL MEGACOLON)

Congenital megacolon was first described by Hirschsprung in 1888. It is a rare condition occurring about once in every 15,000 live births. It is about 10 times more frequent in boys than in girls. A genetic determination is probable since it has been reported in twins and as a familial disorder.

The essential abnormality consists of a very much dilated proximal segment of the descending and sigmoid colon with hypertrophy of the muscle wall and ulceration of the mucosa by the hard faeces. A sharp transition is seen into a narrowed distal portion of rectum which although it appears normal is the site of the trouble. On microscopy this narrowed segment has no parasympathetic ganglion cells and it is believed that, because of this, this piece of bowel is unable to relax to allow faeces to pass. It acts as a partial intestinal obstruction.

*Clinical Features and Course*

Some symptoms are always apparent soon after birth. There may be delay in the passage of meconium. Bile-stained vomiting may occur, associated with abdominal distension and visible peristalsis in the distended large bowel. Rectal examination will release flatus and meconium but constipation persists.

The course is a fluctuating one with periods of apparent temporary improvement, but the infant fails to thrive. The abdomen remains distended, chronic constipation persists and the child becomes anaemic and emaciated. Rectal examination later in infancy always reveals an empty rectum, because of the physiological obstruction higher up. Paradoxically, frequent loose stools may occur in infants who become rapidly ill and dehydrated.

Soiling of the anal region never occurs, nor does faecal incontinence. Without surgical treatment undertaken to excise the distal aganglionic segment of rectum, death occurs in more than 50 per cent of the children by the end of the third year, due to intercurrent infections or to perforation of the distended colon and peritonitis.

*Investigation*

A barium enema by special filling technique is necessary for an accurate diagnosis. In infants a string-like narrowing of the rectum is seen with dilatation of the sigmoid colon and incomplete emptying; in older children there is a less clear-cut narrowing of the rectum with an enormously dilated colon with fluid levels.

*Differential Diagnosis*

Differential diagnosis must be from other causes of intestinal obstruction in infants, with abdominal distension and constipation and from other causes of chronic constipation.

## IDIOPATHIC MEGACOLON (RECTAL INERTIA)

This is a more common condition than Hirschsprung's disease. It is equally frequent in the two sexes and may start at any time during infancy or childhood.

*Clinical Features and Course*

The causes are probably multiple. The results are an inability of the colon *and* rectum (in distinction to Hirschsprung's disease where the rectum is empty) to evacuate themselves. Enormous masses of faeces accumulate in the colon and rectum with dilatation and abdominal distension.

The bowels act infrequently, once a week or at longer intervals. The faeces are hard and dry.

Constipation is usually the presenting symptom, but the child may sometimes be brought for 'diarrhoea'. This is the result of a form of faecal impaction. The fluid faeces are able to seep round the hard masses that remain in the rectum. This seepage together with the

retained masses leads to faecal incontinence and faecal staining of the clothes.

On examination the abdomen is distended and faecal masses can be felt in the descending colon. The anal skin is soiled, the anal sphincter tone is normal and rectal examination reveals a rectum full of faeces.

Unless treatment is undertaken to clear the rectum and colon of faeces to allow the normal urge of defaecation to return the course will be chronic with persistent constipation.

Barium enema must be carried out to differentiate it from Hirschsprung's disease. In idiopathic megacolon the distension of the gut involves the rectum down to the anus.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Newborn* | | | | |
| Physiological | In breast-fed babies bowels may be opened only once a week; this is normal | Clinical | Reassurance | — |
| Imperforate anus | Rare—No meconium passed; deformity may be obvious but in high obstruction appearance may seem to be normal | (1) Clinical<br>(2) Radiograph in inverted position | Urgent surgery | Good with surgery |
| Intestinal obstruction | Rare—No meconium; vomiting and abdominal distension | (1) Clinical<br>(2) Radiograph of abdomen | Urgent surgery and careful follow-up | Good with surgery |
| Hirschsprung's disease | Rare—May be present in infancy with failure to pass meconium | Clinical | Eventual surgery may be required | Outlook doubtful |
| *Infancy* | | | | |
| Physiological | Normal variations | Clinical | Advice | — |
| Underfeeding | Frequent cause | Clinical | Advice | — |
| Anal fissure | Common. Painful defaecation | Clinical | Symptomatic | Good |
| Pyloric stenosis | Rare—Associated with vomiting and pyloric 'tumour' | Clinical | Surgery | Good |
| Mental deficiency | Rare—Constipation is frequent symptom | Clinical | Symptomatic | — |
| Hirschsprung's disease | Rare—Abdominal distension; empty rectum; poor general condition | (1) Clinical<br>(2) Barium enema<br>(3) Rectal biopsy | Eventual surgery | Outlook doubtful |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Childhood* | | | | |
| Physiological | Normal variations | Clinical | Advice | — |
| Emotional | Disturbed mother-child relations | Clinical | Advice | — |
| Symptomatic | Features of primary illnesses such as common fevers, etc. | Clinical | Symptomatic | — |
| Idiopathic megacolon | Rare—Features of persistent chronic constipation; rectum full of faeces; anal soiling | Clinical | Treatment of primary condition | Good |
| Mental deficiency | Rare—Constipation a frequent symptom | Clinical | Symptomatic | — |
| Hirschsprung's disease | Rare—Stunted, pot-bellied child with flat buttocks and wasted limbs, flared ribs and visible peristalsis in grossly enlarged colon; anus clean; rectum empty | (1) Clinical<br>(2) Barium enema<br>(3) Rectal biopsy | Surgery | Poor outlook |
| Neurological causes | Rare—Associated with conditions causing paralysis or interference with anal sphincter | Clinical | Treatment of primary condition | — |

# CONVULSIONS

We are all potential epileptics with varying degrees of natural resistance. At one end of the scale there are those who will convulse only in response to an electric shock or chemical stimulant, while at the other end are those in whom fits seem to occur spontaneously, or rather in response to undetectable stimuli. In between these extremes is a group who respond with fits to stimuli, such as high fever, which will not cause similar reactions in other children.

With such a concept it is obvious that in children in whom the brain tissues are apparently in a more excitable state than in adults there will be a whole variety of possible causes of convulsions.

It has been reported that around 5 per cent of children under 5 years have convulsions at one time or another.

## CAUSES

### NEWBORN

*Birth injuries*—Birth injuries, for example anoxia and cerebral haemorrhage, may cause convulsions at this period or they may be delayed till later in infancy.

*Rare* causes of convulsions in the newborn are:

(1) Congenital cerebral malformations, for instance tuberous sclerosis, amaurotic family idiocy (cerebro-macular degeneration), phenylketonuria, Schilder's disease, porencephaly, cerebral agenesis.

(2) Kernicterus—convulsions are rare, but episodic attacks of opisthotonos and rigidity are common. (See chapter on Jaundice.)

(3) Infections, for instance meningitis, encephalitis, sepsis, toxoplasmosis.

(4) Tetany, which may be due to functional hypoparathyroidism —symptoms occur most commonly at the end of the first week.

### INFANCY

*Main* causes in infancy (6 months to 3 years) are: (1) febrile convulsions, (2) epilepsy.

*Rare* causes are: (1) Infections of the central nervous system, for example, meningitis, encephalitis (especially with common fevers), brain abscess (especially in children with congenital heart lesions).

(2) Degenerations of the central nervous system, for example amaurotic family idiocy, Schilder's disease.

(3) Sequelae of cerebral birth trauma.

(4) Lead poisoning—parents should be specifically asked about pica, especially with regard to ingestion of paint or other lead-containing materials.

### CHILDHOOD

*Main* causes in childhood (after 3 years) are: (1) epilepsy, (2) simple faints.

*Rare* causes are: (1) Space-occupying lesions, for example new growths, abscess, haematoma.

(2) Vascular, for example sinus thrombosis, subdural haematoma, vascular embolism (with congenital heart lesions).

(3) Encephalopathy, in hypertension, uraemia, acute nephritis, whooping cough, toxic states.

(4) Demyelinating states.

## PATHOLOGY

The exact functional disturbances that occur during a convulsion are not completely understood. Basically it is probable that convulsions are caused by periodic and sudden electrical discharges from cortical neurones, but there are three other factors that should be considered.

*Heredity*—A history of fits is found very much more frequently in the families of children with convulsions than in normal families. E.E.G. abnormalities are found in a very high proportion in one or both parents of children suffering from convulsions.

*Disease of the brain*—Convulsions can be a symptom of almost any disorder of the brain. Many of these have been mentioned in the list of causes. However, in many cases of epilepsy and febrile convulsions there are no obvious abnormalities in the brain.

*Precipitating factors*—In children with an inborn or an acquired predisposition convulsions may result from a whole variety of general disturbances, for example high fever at the onset of otitis media, tonsillitis, pyelitis and pneumonia; metabolic upsets—alkalosis, tetany, hypoglycaemia, uraemia and others; cerebral anoxia—in heart lesions, in breath-holding attacks, anaesthesia; and in poisoning with lead.

## DIAGNOSIS

Two problems require consideration in diagnosis. First, it has to be decided whether a convulsion actually occurred. Second, the cause of the attack has to be discovered.

*Was it a Fit?*

Often the doctor arrives on the scene after the episode to find distraught parents and relatives who are unable to give a coherent history.

The doctor's first task is to calm the parents so that an accurate and reliable history may be given. Points in this history which suggest that a convulsion has occurred are a sudden onset, loss of consciousness, tongue biting and incontinence (special attention should be paid to the history of a late onset of enuresis). The suggestion that it might be a 'faint' is usually incorrect. Young children hardly ever faint. Older children (over 10 years) only do so in the erect position on standing (usually at school or in church).

A sign suggestive that a fit has taken place is to find the child rather drowsy and rather quiet and out of contact with the surroundings.

*What Caused the Fit?*

There are three groups of causes: febrile, epileptic and organic convulsions. Helpful differentiating points are as follows:

*History*—In a febrile convulsion there is always a history of fever present before the convulsion. In other types the child may become hot after the attack. Questions should be asked as to the presence of headache, vomiting and ataxia which, if present, suggest a raised intracranial pressure. The possibility of birth injuries should be raised with the parents because the onset of convulsions from this cause may be delayed. They should also be asked whether the child is in the habit of sucking toys and eating the paint. If so, the possibility of lead encephalopathy must be considered. Access to the medicine cupboard and poisoning with a range of drugs must also be considered. A positive family history is the rule in epilepsy but this is also so in a fair proportion (nearly half) of febrile convulsions.

The duration of the attacks varies from a second to some hours. In general febrile attacks last less than 15 minutes.

Convulsions occur at all ages and a list of causes at various periods has already been given. As a general rule fever is the most common

cause up to the age of 3 years, and after that age epilepsy (idiopathic).

*Examination*—A complete physical examination is necessary in all cases of convulsions, but particular attention should be paid to the ears and throat since acute infections of these parts are the most frequent causes of sudden fever. Neck stiffness is suggestive of meningism or meningitis. The fundi should be inspected for papilloedema, haemorrhages and exudates. The limbs should be tested for flaccidity after an attack for if present this suggests an organic cause.

Recurrent attacks are suggestive of epilepsy or organic causes. An E.E.G. examination will be necessary in all recurrent attacks and in all instances where there are suggestions of an organic cause.

More specific tests, such as the blood sugar and blood calcium, may be required on rare occasions.

## SIGNIFICANCE

A convulsion is always a dramatic event of considerable significance. The common causes vary at the different age periods and may be summarized as birth injuries in the newborn, febrile convulsions in infancy, and idiopathic epilepsy in older children.

Other causes, though rare, must always be considered at all these periods before it is decided that expectant management is all that is required.

## SPECIFIC CONDITIONS

### FEBRILE CONVULSIONS

Numerically febrile convulsions are the most frequent type of convulsion in childhood. This is because most fits occur during the first 5 years and at this time it is the febrile convulsion that is the most frequent.

It should be noted that the true febrile convulsion, that is where the high fever is responsible for the fit, is only just one occasion where fits may be associated with fever. Fits resulting from an infection of the nervous system as in meningitis are another example. The energy expended in the fit may itself cause a rise in temperature.

*Aetiology*

The majority of febrile convulsions occur in children aged 6 months to 3 years. Boys are more often affected than girls.

Although an accurate family history is difficult to obtain it is considered probable that in about half of these children there are some relatives who have suffered from fits. This suggests that these children have inherited a brain that is liable to be excited by various cerebral stimuli.

The primary causes of the fever are variable, but acute infections of the ears and throat are the most prevalent.

*Clinical Features*

The convulsions are of the grand mal type. They are of relatively short duration lasting less than 10 or 15 minutes. They are single. Status epilepticus is never the result of febrile convulsions.

Examination of the child will reveal a sick child with a high fever and a search for a cause will usually reveal acute otitis media, acute throat infection, acute bronchitis or more rarely an acute urinary tract infection. An E.E.G. taken a week after the episode is normal.

*Differential Diagnosis*

Although febrile convulsions are the most common type in infants it is always important to bear in mind possible causes that may require urgent and specific treatment, namely meningitis, where there is neck stiffness, the child tends to be much more ill than with a common respiratory infection, drowsiness persists long after recovery from the fit and fits tend to recur. A lumbar puncture and an examination of the cerebrospinal fluid is essential for diagnosis of obscure cases. After an injury, often relatively trivial, and often some time later, drowsiness followed by fits must make the diagnosis of a chronic subdural haematoma a probability.

*Course and Prognosis*

Febrile convulsions are of short duration and apart from possible respiratory obstruction there is little immediate danger. Recurrences are unusual but may occur within 12 or 18 hours after the initial fit.

The ultimate prognosis is good and, providing one can exclude the case of idiopathic epilepsy provoked by fever, there is little likelihood of fits continuing after the age of 5 years.

## EPILEPSY (IDIOPATHIC)

The classification of epilepsy is confusing. The most useful accepted clinical classification is the following:

(1) Major motor, both generalized and focal; (2) minor motor,

including akinetic attacks and myoclonic spasms; (3) petit mal; (4) psychomotor; (5) rare forms.

The importance of epilepsy ranges over many aspects.

*Eugenics*

The predisposition to convulsions is probably inherited as Mendelian recessive. Marriages between first cousins with a positive family history should be avoided, as should marriages between partners who have an epileptic family history. Even where there is only a definite history on one side the risks of the offspring developing epilepsy are much greater than in normal situations.

*General Management*

Once diagnosed, the epileptic patient probably requires management and supervision of some form for life. The epileptic liability of his nervous system is an inherent part of him and attacks may occur in response to stimuli at any time.

The patient in childhood, and more so in later life, requires certain social and personal care. Prevention of injury will require avoidance of potentially dangerous games and activities. Epileptics should not swing, climb, drive motor vehicles or engage in occupations where a sudden fit may place themselves and others in danger.

At the same time every endeavour should be made to treat the child as normally as possible. He should attend normal school if he is sufficiently intelligent. He should mix and take part in all normal activities, providing he is in no danger should a fit occur. His friends and schoolmates should themselves be educated to accept the potential attacks as inevitable and they should be prevented from casting out the affected child as peculiar and undesirable.

*Clinical Features*

The manifestations of epilepsy vary with each type of attack and with the age of the child.

*Major Motor Attacks (Grand Mal)*

In the older child the classical sequence is that of preconvulsive symptoms, such as irritability or moodiness, followed by an aura, and then the epileptiform fit with tonic and clonic features.

In infants there is no such orderly sequence. The first abnormal feature is of generalized twitching which varies in intensity. This is accompanied by frothing of the mouth and cyanosis. The duration of the attack may be a matter of hours.

Recurrent attacks that follow each other occur in status epilepticus. This is a serious complication which is potentially fatal from exhaustion or anoxia.

*Minor Motor Attacks*

These are of two types. In akinetic attacks the child suddenly loses postural tone. Sudden nodding of the head may occur in the sitting position, or if the child is standing he falls to the ground. Myoclonic movements occur in babies and consist of sudden and generalized muscle jerks. These attacks occur in infancy. The outlook is poor as the attacks become replaced by major motor attacks and the children are always mentally retarded.

*Petit Mal*

Petit mal is probably the most frequent form of epilepsy in childhood. It affects children between 3 and 12 years of age. The children are normal in all other respects.

The attacks consist of transient episodes of unconsciousness lasting some seconds. The child stops whatever he is doing, becomes blank and then carries on normally. They may be very frequent—over 100 a day. The E.E.G. is characteristically affected.

The outlook is fair. In the majority the attacks cease at puberty or earlier, but in about a third major motor attacks of epilepsy may occur in adult life.

*Psychomotor Attacks*

These are very rare and are thought to account for a proportion of children with behaviour problems in whom bouts of abnormal behaviour occur for no apparent reason and of which the child has no recollection afterwards.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Newborn* | | | | |
| Birth injuries | Baby affected generally, i.e. listless or irritable; convulsions may be delayed for some time | Clinical | Expectant | Uncertain outcome |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Congenital cerebral malformations | Rare group of conditions including tuberous sclerosis, cerebro-macular degeneration, phenylketonuria, Schilder's disease, porencephaly and cerebral agenesis | (1) Clinical<br>(2) Urine (in phenylketonuria) | Specific in phenylketonuria, non-specific in others | Poor outlook |
| Infections | Rare—Meningitis, encephalitis, sepsis and toxoplasmosis | (1) Clinical<br>(2) C.S.F.<br>(3) Toxoplasmin | Antibiotics where indicated | Fair prognosis |
| Kernicterus | Rare in severe haemolytic disease of newborn | Serum bilirubin high | Treatment as for haemolytic disease | Poor outlook if treatment delayed |
| Tetany | Rare—Symptoms may be delayed for a few days | Serum calcium low | Calcium if indicated | Good prognosis |
| *Infancy* | | | | |
| Febrile convulsions | Common—Associated with primary infection causing high fever | Clinical | Treatment of primary infection | Good prognosis, tends to cease spontaneously after age of 3 years |
| Epilepsy | Recurrent; not associated with fever; family history; various clinical types | (1) Clinical<br>(2) E.E.G. | Treat with anticonvulsants | In majority outlook is good; few may present social problems |
| Infections | Rare—for example meningitis, encephalitis and brain abscess | (1) Clinical<br>(2) C.S.F. | Treat infection | Prognosis uncertain |
| Degenerations | Rare—For example cerebro-macular degeneration and Schilder's disease | Clinical | Non-specific | Bad prognosis |
| Sequelae of birth trauma | Rare—Convulsions may be delayed until child is a few months old | Clinical | Non-specific | Poor prognosis |
| Lead poisoning | Rare—Should be considered in all cases of doubt | (1) Clinical<br>(2) Lead content of blood | (1) General<br>(2) Chelating agents | Good prognosis in most cases |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Children* | | | | |
| Epilepsy | Commonest cause of convulsions; various clinical types | (1) Clinical<br>(2) E.E.G. | Anticonvulsants | Good prognosis in majority |
| Faints | Uncommon in children; convulsions due to anoxia | Clinical | Symptomatic | Good |
| Space-occupying lesions | For example new growths, abscess, blood clot | Specific for disease | That of primary cause | Depends on cause |
| Vascular | For example subdural haematoma, thrombosis, embolism and acute infantile hemiplegia | Specific for disease | That of primary cause | Depends on cause |
| Encephalopathy | For example hypertension, nephritis, uraemia, diabetes mellitus, whooping cough | Specific for disease | That of primary cause | Depends on cause |

# COUGH

The child with a cough is one of the most frequent reasons for seeking medical advice in general and paediatric practice.

Quite apart from a wide range of possible physical and organic causes it is possible that the reason for seeking advice is the mother's anxiety and personal emotional disturbance.

## CAUSES

A simple aetiological classification is not possible and it is most practical to consider causes in the upper and lower parts of the respiratory tract (in spite of its unity) and to note which are common and which are rare conditions.

### UPPER RESPIRATORY TRACT

*Common*

*The common cold*, as an acute infective illness accompanied by a running nose and possibly by fever, is probably the most frequent cause of a cough. It may be of short duration or it may be the start of a long period of recurrent respiratory ailments.

*Influenza* is a specific epidemic illness. Its main features are a sudden onset, fever and malaise and a dry irritating cough. Diagnosis is most likely to be accurate during an epidemic. Sporadic cases of 'flu' are much more likely to be due to other causes.

'*Catarrhal children*' frequently have chronic and persistent coughs. Causes include sinusitis, chronic bronchitis and enlargement and infection of the tonsils and adenoids.

*Laryngitis* in its acute form causes a characteristic tight, croaking and croupy cough. (See chapter on Croup.)

*Rare*

*Ear*—Irritation of the auricular branch of the vagus nerve (Arnold's nerve) in the external auditory meatus by an impacted foreign body or dry and hard wax may rarely cause an irritating and useless cough.

*Retropharyngeal abscess* in its subacute and chronic forms is a very rare cause of an irritating cough.

LOWER RESPIRATORY TRACT

*Common*

*Acute bronchitis* is a frequent condition of children—nearly 10 per cent will suffer an attack at some time. It is recognized by evidence of systemic infection and diffuse and bilateral abnormal chest signs.

*Pneumonia* is differentiated from acute bronchitis chiefly by the greater severity of the illness.

*Measles*—The earliest symptom of measles is the dry, irritating, persistent and distressing cough. The rash appears some 3–4 days later.

*Whooping cough* is characterized by paroxysms of coughing with an inspiratory whoop and vomiting.

*Asthma*—Short sharp coughs with stringy mucus accompany tight wheezing during asthmatic attacks. (See chapter on Wheezing.)

*Emotional disturbances* in the child or the parents may aggravate any of the conditions causing cough.

*Rare*

*Tuberculosis* is now a rare infection in British children. It must, however, be considered and differentiated in children with a persistent cough and that enlargement of the mediastinal glands seen on radiograph, which forms part of the primary complex. The adult type of pulmonary tuberculosis is excessively rare.

*Foreign* bodies may sometimes be inhaled without any history being available to the doctor. This possibility should always be considered in chronic and unexplained lung infections. A bronchoscopy is necessary in such situations.

*Collapse* of lobes or segments of the lung may result from obstruction of bronchi. This may follow conditions associated with sticky mucus as in whooping cough and asthma, or from pressure of glands or growths outside the bronchi.

*Bronchiectasis* follows collapse. It is rare and is associated with expectoration of much infected sputum.

*Lung abscess* may follow bronchial obstruction or severe pneumonia.

*Enlarged mediastinal* glands or mediastinal tumours will cause an irritating cough from local pressure.

*Fibrocystic disease of the pancreas* is associated with a liability to recurrent respiratory infections. The cough at times is paroxysmal and resembles whooping cough.

*Hypogamma-globulinaemia* may also cause recurrent respiratory infections because of the low state of immunity of the affected child.

## PATHOLOGICAL DISTURBANCES

*Cough* is an important physiological defence mechanism. It serves to protect the lower respiratory tract from infected secretions, food and foreign bodies from the mouth, nose and throat and also removes secretions and exudates from the bronchi and bronchioles. In children these exudates mixed with mucus are coughed up into the mouth and swallowed. It is rare for sputum to be expectorated unless it is produced in great amounts, as in bronchiectasis. When present for inspection sputum usually suggests the presence of serious local disease.

The relatively narrow calibre of the bronchi and bronchioles predisposes to obstruction of these tubes, especially when the cough reflex is diminished or absent, as it may be in infants during sleep and anaesthesia.

The centre for the cough reflex is sited in the medulla of the brain, close to the vomiting centre. It receives afferent impulses from the lining mucosa of the respiratory tract through the vagus and glosso-pharyngeal nerves.

The act of coughing begins with a short inspiratory phase followed by closure of the glottis and the onset of a forceful expiration. With the opening of the glottis there is a sudden release of pressure and the child coughs, expelling any accumulated secretions or exudates.

The basic causes of the cough are irritation of the sensitive respiratory mucosa. This occurs from inflammations caused by a variety of organisms in a number of possible conditions and from irritation resulting from mechanical causes such as foreign bodies, pressure from masses and the accumulation of excessive secretions as in the allergic reaction in asthma.

## DIAGNOSIS

The elucidation of the diagnosis in a child with a cough is largely a clinical exercise.

*History and Examination*

The history should elicit information on contact with persons with possible infectious illnesses such as measles and whooping cough. The child's previous history will enable a decision on whether

he (or she) is passing through the 'catarrhal' phase when there is an increased likelihood of recurrent respiratory infections. Recurrent wheezy attacks suggest the possibility of asthma and here a history of infantile asthma and a positive family history are further diagnostic points.

The character of the cough is helpful. A dry, hacking and unproductive cough is associated with an irritation of the upper respiratory tract from excessive nasal mucus and post-nasal obstruction, but it may also be a feature of early measles, mediastinal pressure or an inhaled foreign body.

A croaky seal-like cough that is worse at night is almost pathognomonic of an acute laryngitis.

A 'juicy', loose and productive cough suggests involvement of the lower respiratory tract, as does associated wheezing.

Reference has been made to whooping cough with its paroxysms, whoop and vomiting. This type of cough may occur also in children with fibrocystic disease and in some with an inhaled foreign body that is capable of some movement within the bronchus.

Examination should be general and include the whole child but with particular reference to the ears, nose and throat, as well as the chest.

*Ancillary Investigations*

A chest x-ray should be carried out in children in whom the cough has persisted for longer than a few weeks, or less if there are any more definite indications. Although this x-ray will be normal in the majority it may serve to demonstrate collapsed lobes, segmental or lobular infections and enlarged mediastinal glands. Children with such lesions require more careful management and supervision.

A tuberculin (1/1,000) skin test should be carried out where the child continues to be vaguely ill, with general malaise, anorexia and cough.

Bronchoscopy is indicated in children with persistently collapsed lobes, in cases of haemoptysis, when there is a suspicion of an inhaled foreign body and when it is necessary on very rare occasions to obtain bronchial secretions for examination in possible bacterial, tuberculous or mycotic infections.

In children with severe recurrent respiratory infections the possibilities of fibrocystic disease of the pancreas and hypogammaglobulinaemia should be remembered and stools tested for excess of undigested fats and proteins, the sweat for excess of sodium chloride and the blood for low levels of *gamma*-globulin.

## SIGNIFICANCE

Faced with very many children suffering from coughs which they may eventually outgrow it is all too easy for the doctor to miss the rare case where there is some more serious underlying condition.

The following are situations when he should pause and think twice before passing it off as 'a common non-specific respiratory infection'.

(1) In infants a cough is always of significance.

(2) When a cough persists for longer than a month then further investigations are necessary.

(3) When sputum is available for inspection then it is very likely that there is some serious underlying local condition present such as bronchiectasis, lung abscess, infected collapsed lobe, fibrocystic disease or an inhaled foreign body.

(4) Cough associated with progressive or persistent general signs such as loss of weight or anaemia or other abnormal signs must always be carefully investigated.

## SPECIFIC CONDITIONS

### 'CATARRHAL CHILDREN'

There is a definite syndrome in children between 3 and 8 years of age, when they pass through a phase with frequent respiratory tract infections. These infections may take the form of colds, coughs, earaches, sore throats or more specific illnesses as acute bronchitis and pneumonia. Because of the unity of the respiratory tract these diverse conditions are all closely related.

*Epidemiology*

*Frequency*—The frequency of the condition is such that scarcely any child escapes at least one episode annually. Some suffer continually over a number of years from one or other clinical variety of the syndrome. Boys are more likely to be affected than girls.

*Season*—The incidence begins to increase from October and the peak is reached in January and February.

*Social class*—This syndrome is no respecter of persons. The frequency is roughly similar in upper and lower classes, but the infections are more severe with many more complications in the lower social groups. The peak incidence occurs earlier, at about 2–4 years of age, in lower social groups, whereas this is delayed until 6–8 years of age in the upper classes. This presumably is related to the

earlier social mixing with infective agents that occurs in the lower social groups. These common infections also occur earlier in younger siblings because they are brought home by the older brothers and sisters.

*Clinical Features*

Whatever the main feature might be—earache, sore throat, bronchitis or nasal obstruction—cough is a frequent symptom.

The cough, an irritating one, is present during the day, but it is worse at night.

Examination does not usually reveal any dramatic abnormalities. The nose is stuffy, the tonsils congested, and the chest clear.

*Associated Features*

The cough which may be the presenting sign is only one feature of this syndrome. Infections of the ears and throat are frequent.

*Course*

The course is characteristic. It seems that the affected child passes through a couple of winters when the features of the syndrome are at their peak. An apparent resistance is then built up and there is little further trouble from respiratory infections. This peak occurs between 4 and 7 years of age in most children, but it may be earlier or later depending on the individual circumstances already referred to.

*Management*

Management of these children is often trying and difficult. It must be based on good relations and understanding between mother and doctor, on judicious use of antibiotics, on selective use of surgical procedures such as tonsillectomy and adenoidectomy and above all on treating the problem as a whole and appreciating that the natural tendency is for the child to outgrow the syndrome.

## ACUTE BRONCHITIS

Acute bronchitis is a frequent illness in childhood. Nearly one child in every 10 will suffer from acute bronchitis or pneumonia at some time during his first years of life.

*Clinical Features*

The clinical features of acute bronchitis are those of fever and malaise together with a tight cough and often associated with wheezing and breathlessness. The physical signs suggest a diffuse and

bilateral involvement of the lower respiratory tract. Wheezy rhonchi and moist rales can be heard over both sides of the chest but are maximal in the lower zones.

The condition most often follows an acute upper respiratory infection. It may, however, be associated with measles and whooping cough.

*Complications*

In those under 2 years of age convulsions are not uncommon. Dehydration is a danger when there is high fever and when diarrhoea and vomiting are present.

*Epidemiology*

Epidemiologically acute bronchitis occurs most often in infants in the first years of life, and in children in their first two years at school. Attacks recur in about 10 per cent of children.

The tendency is for most children to outgrow these attacks of bronchitis but in a very small proportion the liability to recurrent bronchitis persists and the child will eventually develop into an adult chronic bronchitic.

### PNEUMONIA

The differentiation between pneumonia and acute bronchitis is essentially one of degree. Pneumonia may be localized or diffuse. It may be caused by a variety of organisms and other factors. The most important organisms are pneumococci, *Haemophilus influenzae*, haemolytic streptococci, staphylococci and viruses. Other aetiological factors may be aspiration of infected mucus or food.

*Clinical Features*

The onset is sudden, with high fever and prostration. A convulsion may be the first sign in infants. Cough is prominent. The cough is short and sharp and distressing to the severely ill child. There may be pain—which is sometimes referred to the abdomen. Herpes labialis may develop on the lips after a few days of illness.

On examination the child is restless yet still. One look reveals that the illness is a major one with rapid breathing and usually an expiratory grunt. The accessory muscles of respiration are in action. Abnormal physical signs may not be obvious at onset and a presumptive clinical diagnosis may have to be made. Within 48 hours crackling rales will be heard either all over the chest or more often at one or other base.

*Complications*

These include convulsions, meningism (especially with an upper lobe pneumonia), empyema, and sudden tension pneumothorax (with staphylococcal pneumonia).

*Differential Diagnosis*

An acute abdominal emergency has to be considered in a suddenly severely ill child with fever, vomiting and abdominal pain. Points suggesting an abdominal lesion are a low fever, vomiting—almost invariable in appendicitis and other abdominal emergencies—furred tongue, guarding and tenderness in the lower abdomen.

Pyelitis may present with a picture of a feverish sick child, usually a girl. There is usually tenderness in the loins and the urine always contains pus cells and bacteria.

Meningitis must be considered where there is vomiting, drowsiness, fever and neck stiffness.

*Staphylococcal Pneumonia*

This requires a special note. It occurs most frequently in infants. Usually associated with some preceding staphylococcal lesions such as boils, septic spots or infected wounds, it is relatively common in children suffering from fibrocystic disease. It can only be diagnosed with certainty by recovering the causal organism, but this may be very difficult in infants.

## BRONCHIECTASIS

With better and effective treatment of acute chest infections bronchiectasis is becoming rare.

The majority of cases are diagnosed between 3 and 10 years of age, although the causal infection may have occurred some time before the diagnosis is made.

Like most serious chest conditions bronchiectasis is found much more frequently in children belonging to the lower social groups.

*Aetiology*

The two main aetiological factors are severe infection with destruction of bronchial mucosa, and bronchial obstruction and collapse of the distal part of the lung. These factors are present in severe pneumonias, whooping cough, aspirated foreign bodies and rarely (now) in tuberculosis.

*Clinical Features*

The clinical features appear gradually. A persistent 'juicy' cough

is the main feature. This is usually present for many months before the diagnosis is made, or even discovered. Although present during most of the day the cough is worse in the morning. Sputum is often present and can be collected for inspection by the doctor: this is almost pathognomonic (in no other condition in childhood can quantities of sputum be collected). It is usually yellow or green. In early cases there is little interference with growth and development and the only abnormal signs are persistent moist rales at one, or both, bases. In later stages deformities of the chest may occur, with finger and toe clubbing.

Chest x-ray may show nothing abnormal. A bronchogram is required to confirm the diagnosis and localize the extent of the disease. A bronchoscopy should be performed to rule out the possibility of an unsuspected foreign body or some other cause of bronchial obstruction.

### WHOOPING COUGH

Routine immunization against whooping cough has reduced both the severity and the incidence of the disease. Nevertheless it still occurs and is potentially serious and dangerous in infants. Immunization in infants should be started by the second month.

It is an infection of the respiratory tract caused by *Haemophilus pertussis* or *H. parapertussis*. Transmission is by direct droplet infection. Affected children are most infectious in the pre-whoop catarrhal phase and remain so for about 4 weeks.

*Clinical Features*

Clinically the onset is insidious with a short-sharp, dry cough, worse at night. The cough increases over a few weeks and becomes paroxysmal, occurring in definite attacks that tend to be worse at night. Each attack begins with a series of short sharp coughs that end in a deep inspiratory whoop with holding of breath. The attacks are exhausting and end with expectoration of thick, sticky mucus that is often vomited up. Improvement occurs after 5–6 weeks.

*Complications*

Complications may be local pneumonic infection, blockage of bronchi and lobular or lobar collapse of the lung.

More distant lesions may also occur in the central nervous system with convulsions, transient paralyses and encephalitis. Mechanically, severe paroxysms may cause oedema of the face, surgical emphysema of the neck, subconjunctival haemorrhage, nose-bleeds and herniae.

*Differential Diagnosis*

In differential diagnosis two causes of paroxysmal cough should be remembered—fibrocystic disease and obstruction of the trachea, either from foreign bodies or from external pressure from lymph glands.

## MEASLES

Measles is a well-nigh inevitable infection of childhood. Most commonly contracted by the child in the first two years of school life, it is then passed on to any younger siblings at home.

It is the most infectious illness in children and is spread by direct contact–droplet infection having an incubation period of 12–16 days.

*Clinical Features*

In most children the first clinical sign is an acute coryzal illness with a very irritating cough as the main feature. The prodromal phase accompanied by intense misery and red eyes is followed by the appearance of the characteristic macular rash after 2–4 days. Rarely, this prodromal phase may persist for as long as 10–14 days and this leads to much difficulty with diagnosis. The well known Koplik's spots are obvious in no more than half of all children and the early diagnosis should not be ruled out because of their absence.

*Course*

The usual course is for the prodromal phase to last 2–4 days and the rash to persist for 4–6 days. Full recovery should be anticipated within 7 days of the onset.

*Complications*

Complications are now rare. Otitis media and pneumonia are the most frequent, but encephalomyelitis and convulsions may occur.

## FIBROCYSTIC DISEASE (MUCOVISCIDOSIS)

This rare familial disease may present with the picture of a child with a persistent and troublesome cough. Its incidence is about 1 per 1,000, so that an average-size general practice in Great Britain will possibly have one or two cases.

*Clinical Features*

Clinically varying degrees of severity exist with different presentations depending on the target organ affected.

Where the *lungs* are affected the symptoms in severe cases are present from birth. In these less severely affected the condition will not become apparent until the child is 2 or 3 years old. Recurrent attacks of 'bronchitis' are usual. The cough tends to be spasmodic in character and resembles whooping cough. Large amounts of infected sputum will be expectorated.

Staphylococcal pneumonia is a frequent complication and if the child lives long enough bronchiectasis may develop.

In other children intestinal symptoms may predominate. Owing to undigested fats, the stools are bulky, fatty and frothy.

*Diagnosis*

The diagnosis is confirmed by finding trypsin to be absent from the duodenal juice, by the steatorrhoea and by demonstrating an excessive amount of sodium chloride in the sweat.

*Prognosis*

The outlook is very poor, although now, with long-term antibiotics to control respiratory infections, the child may reach adolescence and even adult life.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Common cold | Acute onset; little systemic upset | Clinical | Symptomatic | |
| Influenza | Epidemic illness; acute onset; considerable systemic upset | Clinical; diagnose only during an epidemic | (1) Symptomatic<br>(2) Antibiotics for complications | |
| Catarrhal children | Syndrome of recurrent respiratory infections causing nasal obstruction, otitis media, sore throats, chest infections | Clinical | (1) Symptomatic<br>(2) Antibiotics<br>(3) Tonsillectomy and adenoidectomy (rarely) | Tendency for natural improvement at 2–8 years of age |
| Laryngitis | Acute and often recurrent; croaky cough; may be complicated by respiratory obstruction | Clinical | (1) Symptomatic<br>(2) Rarely require admission for respiratory obstruction | Tendency to recur; tendency for natural improvement after 3 years of age |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Acute bronchitis | Acute and recurrent variable systemic upset; diffuse signs in chest; wheezy | Clinical | Antibiotics | Tendency to recur; tendency to natural improvement; good response to antibiotics |
| Pneumonia | Acute; severe systemic upset; abnormal chest signs | Clinical and radiograph of chest | Antibiotics | Good response to antibiotics |
| Measles | Cough, red eyes and coryza at onset; Koplik's spots; rash after 2–4 days | Clinical | (1) Symptomatic<br>(2) Antibiotics for complications | Now a relatively mild illness; few complications |
| Whooping cough | Characteristic paroxysmal cough with whoop and vomit | (1) Clinical<br>(2) Coughplate | (1) Symptomatic<br>(2) Antibiotics for complications | Serious in infants. Good protection by immunization |
| Asthma | Recurrent paroxysmal attacks of wheezy dyspnoea; frequent association with eczema and positive family history | Clinical | (1) Bronchodilators<br>(2) Steroids (rarely) | Tendency to improve with age |

# CROUP

The old term 'croup' is useful to describe a group of conditions with stridor and laryngeal obstruction as the main features.

## CAUSES

### ACUTE INFECTIONS

(1) Spasmodic laryngitis: by far the most frequent cause of stridor.

(2) Laryngo-tracheo-bronchitis: a serious and potentially fatal condition in infants.

*Rare*

(1) Retropharyngeal abscess: obstruction of larynx by external pressure of the swelling.

(2) Diphtheria: obstruction caused by membrane and oedema.

### CONGENITAL

Congenital laryngeal stridor: a not uncommon cause due to flaccidity of epiglottis and adjacent tissues.

*Rare*

(1) Laryngeal web: mucous membrane web extending across anterior part of vocal cords and glottis; marked respiratory obstruction; absence of a cry.

(2) Cysts: laryngeal or thyroglossal.

(3) Stenosis: subglottic or tracheal.

### LARYNGEAL OEDEMA

(1) Allergy: angioneurotic oedema; anaphylaxis after injections of serum or penicillin; wasp or bee stings.

(2) Trauma: indelicate bronchoscopy or tracheal aspiration; after operation in which an endotracheal tube was used; inhaled foreign bodies; dislocation of crico-thyroid or crico-arytenoid joints; inhaled or swallowed irritants.

(3) Infections: spread from infections in the neck and throat.

### LARYNGEAL SPASM

Trauma: during intubation of larynx, during anaesthesia.

*Rare*

Spasmophilia (laryngismus stridulus) in rickets and tetany.

NEOPLASMS

Laryngeal papillomas: appear in infancy and disappear at puberty.

*Rare*

Extrinsic: mediastinal masses, cystic hygroma, thyroid tumours, lymphomas and thymus enlargements.

## PATHOLOGY

The essential disturbance in croup and stridor is an obstruction of the airway at the level of the larynx.

The causes of this obstruction may be swelling due to infections, allergy and trauma; spasm from traumatic irritation and from over-excitability as in tetany and rickets; and mechanical factors such as foreign bodies, new growths and diphtheritic membranes.

The effects of the obstruction are to cause anoxia with eventual suffocation if unrelieved.

## DIAGNOSIS

Stridor is a harsh, vibratory and high-pitched crowing noise most marked during inspiration. It is an important sign of respiratory obstruction.

Clinical manifestations include hoarseness, breathlessness, inspiratory retraction of the chest wall, use of accessory muscles of respiration, brassy cough, fever and raised pulse rate. Restlessness is an early sign of anoxia and is present before any obvious cyanosis.

An aetiological diagnosis can usually be made on clinical grounds by careful attention to the history and the presenting signs.

The age is important. Congenital causes will be present soon after birth. Acute infections are most frequent between 6 months and 3 years of age.

The history will often help to establish an allergic or traumatic experience and in acute spasmodic laryngitis there is a definite tendency to recurrent attacks.

Ancillary methods of investigation are of little practical use.

Direct laryngoscopy by an expert is indicated if the cause of obstruction cannot be explained easily; but there is a very real danger of reactive oedema following this procedure.

## SIGNIFICANCE

Croup or stridor is always a potentially dangerous symptom and the child should be seen and examined by the doctor as soon as possible, to determine the cause and assess the general condition. In laryngo-tracheo-bronchitis the course may be very rapid indeed and the child may be dead within 6–12 hours from the onset.

Increasing restlessness is a serious sign and an indication for admission to hospital and possible tracheostomy. The dangers of laryngeal obstruction are much greater in infants. This is because of the small size of the larynx, and the loose connections and folds in the region of the glottis which may collapse and obstruct the airway.

## SPECIFIC CONDITIONS

### ACUTE SPASMODIC LARYNGITIS (LARYNGITIS STRIDULOSA)

This is the most frequent cause of 'croup' in children. It occurs more often in boys than in girls and generally in the 2-5 years age group. There seems to be a definite individual predisposition which is very often a familial trait. These children are rather nervous and highly strung and inclined to be overweight. Attacks occur chiefly in the winter months following upon an upper respiratory infection.

*Clinical Features and Course*

A child suffering from a cold and cough goes to bed off-colour but not obviously ill. The child awakens in a cold bedroom on a cold winter's night in the early hours of the morning distressed, fighting for breath, with a most distressing barking cough and hoarseness. An inspiratory stridor is present and there is indrawing of the intercostal spaces and supraclavicular fossae. The parents are terrified (especially in the first attack, and this makes the child's condition worse).

With suitable simple remedies, such as taking the child into a warm room, and giving hot drinks, steam inhalations and sedatives, the attack will pass off, only to recur later in the illness or on a future occasion.

The course is almost always benign, the acute condition settling

within a few hours. It is most unusual for attacks to occur after the age of 5 years.

### ACUTE LARYNGO-TRACHEO-BRONCHITIS

Potentially this is a very serious condition with an acute rapidly descending infection of the respiratory tract. It is caused by viruses with secondary bacterial invaders.

Fortunately it is rare. Cases tend to occur in small epidemics during winter months. Children between 3 months and 3 years of age seem most susceptible.

Pathologically there is an acute and intense inflammatory reaction in the pharynx, epiglottis, larynx and trachea with spread to the bronchi and bronchioles. Much sticky mucus is produced and this tends to occlude and obstruct the small airways.

*Clinical Features, Course and Management*

The onset may be very sudden with acute respiratory distress and collapse out of proportion to the degree of anoxia. The child is shocked and clammy, with a low fever. Stillness rather than restlessness is a serious feature in these children. Evidence of severe respiratory obstruction is obvious. All the accessory respiratory muscles are in action, the intercostal spaces indrawn and there is noisy breathing with a marked inspiratory stridor.

The course may be very rapid. Death may occur within a few hours.

Management must be based on continuous and careful observation with expert nursing, maintenance of a free airway and antibiotic therapy in hospital.

### CONGENITAL LARYNGEAL STRIDOR

This is not a specific disorder but rather a syndrome comprising a number of possibilities. It is not uncommon and may be familial.

Symptoms generally appear initially in the first days of life but may not do so until a month or two later.

Most cases of 'inspiratory laryngeal collapse syndrome' present with a crowing noise on inspiration, expiration being normal. This noise is more striking when the baby cries or becomes excited.

Although less marked during the night and when asleep, the crowing is present continually. Respiratory distress is absent and there is no interference with feeding. This condition begins to clear up spontaneously from one year of age and disappears altogether by

the age of 5 years. The cause is an anatomical one due to an exaggeration of certain features of the normal infantile larynx. The aryepiglottic folds are over loose and tend to collapse on inspiration with the consequent crowing sound. There is no treatment for this condition but a laryngoscopy is necessary to exclude other much less common causes such as cysts and haemangiomas. In these there is always difficulty with breathing and bouts of cyanosis occur.

### LARYNGISMUS STRIDULUS

This is now almost unknown. It was part of the classical triad of rickets that affected the nervous system and consisted of convulsions, spasmophilia and laryngismus stridulus. It was characterized by intermittent sudden attacks of stridor that lasted for only a few breaths and were followed quickly by normal breathing.

### LARYNGEAL PAPILLOMAS

Laryngeal papillomas are more often a cause of hoarseness and aphonia than of stridor, although this can occur in more severe obstruction. Boys are affected, from infancy upwards. Recurrence after removal is common but there is a natural tendency for them to disappear at puberty. Malignant changes do not occur.

### TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Acute spasmodic laryngitis | Acute onset at night; recurrent; age 1–5; males predominate; benign | Clinical | Symptomatic | Recurrent but rare after 5 years of age |
| Laryngo-tracheo-bronchitis | Acute or insidious onset; may spread rapidly to bronchioles causing death; restlessness a sign of anoxia | Clinical | (1) Antibiotics<br>(2) Admission to hospital<br>(3) Tracheostomy may be necessary | Appreciable (5–10 per cent) mortality rate |
| Congenital laryngeal stridor | Present soon after birth; inspiratory stridor; benign | (1) Clinical<br>(2) Laryngoscopy (in some cases) | No special therapy | Clears up spontaneously by age of 5 years |
| Oedema of larynx | Secondary to other conditions such as allergies, trauma and infections | Clinical | May be an emergency requiring tracheostomy | Death may occur in severe cases of obstruction if un relieved |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Spasm of larynx | Secondary to surgical interferences, as intubation, bronchoscopy, and to tetany and rickets | Clinical | May require tracheostomy | |
| Laryngeal papillomas | Appear in infancy; hoarseness main feature | Laryngoscopy | Surgical removal may be necessary | Recur after removal but disappear after puberty |

# DEAFNESS

The early recognition of deafness is a matter of prime importance in order that the development of the child should not be interfered with.

## CAUSES

Traditionally deafness is classified as:

*Conductive*, when the cause is in the outer or middle ear.

*Perceptive*, when the cause is in the inner ear or in the auditory nerve.

*Central*, when the cause is in the higher nervous centres.

A much more practical grouping of causes is one based on the age at which deafness becomes recognizable.

### CONGENITAL

These are all rare forms of perceptive deafness.

*Infections*

*Maternal*, such as rubella (german measles) causing damage to the inner ear.

*Foetal*, congenital syphilis, causing damage to the inner ear and the nervous system.

*Other Causes*

*Kernicterus*, associated with severe jaundice in haemolytic disease of the newborn. (See chapter on Jaundice.)

*Genetic*, a rare form of inherited abnormality, that is Mendelian and recessive.

No obvious cause can be found in many cases of congenital deafness.

### INFANCY

*Infections*

*Otitis media*, acute, recurrent and chronic, causing conductive deafness. (See chapters on Earache and Ear Discharge.)

*Meningitis*, tuberculous, meningococcal, pneumococcal and staphylococcal, causing damage to the auditory nerve, and the inner ear. (See chapters on Coma and Headache.)

*Streptomycin*

Streptomycin used therapeutically in tuberculous infections may cause damage to the inner ear and the nerve tracts.

### CHILDHOOD

*Wax* in the external meatus is a common cause. Less frequent is an impacted foreign body.

*Otitis media*—acute, recurrent or chronic.

*Eustachian tube obstruction* from enlarged adenoids or chronic post-nasal infections.

*Encephalitis* associated with mumps or other common fevers may cause damage to the auditory nerve. (See chapter on Headache.)

*Trauma* such as fractures of the base of the skull may damage the inner ear or auditory nerve.

## PATHOLOGY

Sound waves are vibrations of the air. A range of from 32 to 20,000 cycles per second is audible to the human ear. Speech covers from 256–2,048 cycles per second.

Sounds are concentrated by the pinna, but this has a relatively minor function in man. They are then transmitted along the external meatus to the membrane of the ear drum. This is caused to vibrate, and its vibrations move the three ossicles of the middle ear, the malleus, incus and stapes. The foot-plate of the stapes is in contact with the inner ear, the cochlea. This cochlea has a most sensitive nervous receptive mechanism and from it impulses are sent along the auditory nerve to centres in the medulla of the brain and finally to the cerebral cortex.

### ANATOMICAL LEVEL OF INTERFERENCE

Interference with this fine and delicate mechanism can occur at any level. In the meatus obstruction can be caused by wax, debris or foreign bodies. In the middle ear infections can cause permanent damage to the ear drum and the ossicles. Obstruction of the eustachian tubes, whose function is to maintain a constant atmospheric pressure in the middle ear, will interfere with transmission of sounds. In the inner ear damage to the cochlea can result from trauma and infections. The nerve tracts, that is the auditory nerve and intracerebral tracts, can also be damaged by infection and trauma.

## PRESENTING SYMPTOMS IN CHILDHOOD

### DEVELOPMENT AND HEARING

Hearing is necessary for normal development in children. By the age of 3 months some appreciation of sound should be evident; by 6 months the child should turn his head in response to noise and attempts should be made to vocalize in response to speech; by 12 months he should recognize the sound of his name and say a few words; by the age of 2 years he should be able to say a few sentences.

Interference with normal hearing leads to a failure in the normal coordinated development of the child. The deaf child will tend to appear backward and behave in a backward fashion. This will occur even with relatively slight degrees of deafness which, because they are more frequent than complete deafness, are therefore all the more important.

Early recognition is very important both for preventive and therapeutic reasons. If the diagnosis is missed the deaf child is very liable to be labelled as 'backward', with all its implications. Deafness is an important factor in causing behaviour problems, with temper tantrums, bouts of screaming and accusations of disobedience, inattentiveness and lack of interest at school. The development of speech is also interfered with. Children with complete deafness during their first two years of life are dumb because they have never been able to hear spoken words. They are deaf and dumb because they have never had the opportunity to learn and practise speech. Partial deafness will cause speech defects because there is a distortion of the sound pattern reaching consciousness.

## DIAGNOSIS

The diagnosis of deafness in early childhood is difficult. Awareness of its possibility is important in children with any problems of development. Particular attention must be paid to the mother's observations. She should not be fobbed off, ridiculed and wrongly reassured without suitable tests being carried out. Even if these appear normal, careful follow-up and observation with repetition of the tests must be organized.

It is rare for the diagnosis to be made before 6 months of age, but suspicions must be followed up and expert advice sought whenever doubt exists.

### TESTS FOR DEAFNESS

*Infancy*

*Hearing response*—The baby may not respond to any noise at all, or

may respond only to loud noises and not to the mother's voice. All normal babies respond to their mother's voice by 6 months of age.

*A completely deaf child will not talk*—He will cry more than usual in a particularly screeching manner. The partially deaf child when talking will do so in a dull and monotonous fashion.

*To compensate for lack of hearing visual reactions become much more acute than usual*—He tends to be attentive and use gestures a great deal.

*Emotional liability*—Temper tantrums are frequent. Mood changes from irritability to sullen quietness. Both reactions tend to be one of the results of a lack of understanding and of being misunderstood.

*Later Childhood*

In later childhood certain specific tests can be carried out to test for deafness which may present as a behaviour problem at home or at school or as speech defects. These tests are best left to the expert.

*Distraction techniques*—These can be used in children between 6 months and 2 years. While the child's attention is attracted a loud sudden noise is made behind him. A deaf child will remain unmoved, a normal one will immediately turn in the direction of the sound.

*Conditioning techniques (2–3 years)*—These depend on first conditioning the child to perform a simple act in response to simultaneous combined visual and auditory stimuli. Once this conditioned act has been learnt the effects of using the auditory stimulus alone are noted. In the completely deaf there will be no response. In those partially deaf some response may occur.

*Audiometry (over 3 years)*—This is the ideal method of measuring hearing, but it is open to many errors when carried out by the inexpert. Audiometric records are particularly useful when repeated over a period to assess progress or to confirm previous records.

## SIGNIFICANCE

Deafness as a symptom must always be taken very seriously. Investigations must be carried out to establish whether the child is in fact deaf, and if so, why. A decision has then to be made as to the correct management of the situation. That must always include the family as well as the individual child.

*Is Deafness Present?*

The younger the child, the more difficult are the techniques required to establish whether the child is deaf and what is the extent of the deafness.

### PRESENTING SYMPTOMS IN CHILDHOOD

A deaf child only rarely complains of 'deafness'. A general disturbance is the most usual representation—slow development in infancy, failure to talk or speech defects, behaviour problems and failure to progress at school. Deafness should always be considered as a possibility in these circumstances.

*What are the Causes?*

The causes of deafness can usually be determined clinically. Conductive deafness due to disorders of the external and middle ear can be improved or corrected by suitable therapy and it is important to pick out these children as soon as possible.

*What Should be Done?*

In many deaf children there is no specific cure but they can always be helped considerably by correct management.

It is essential to have an expert's opinion as soon as possible to confirm that deafness is present and whether there is any specific therapy available.

Consideration must also be given to the future management. Here again expert advice is necessary. It may be best for the child to be admitted to a residential home or school at the start. All efforts must be made to make the best use of whatever hearing there is by means of hearing aids, and the child's visual and other sensations must be developed to help the child to make and keep contact with the outside world.

## SPECIFIC CONDITIONS

### CONGENITAL DEAFNESS

This is an uncommon type of perceptive deafness due to causes within the inner ear or in the nerve tracts. Partial deafness is more frequent than complete deafness. Particular awareness must be exercised when there is a maternal history of rubella during the early weeks of pregnancy or of an Rh negative blood group in the mother, or in prematurity and in association with other congenital abnormalities such as mongolism and congenital heart lesions.

In making the diagnosis attention must be paid to the developmental history and the ages at which the 'milestones' have been passed.

The diagnosis should always be confirmed by an expert. Much time and patience are needed.

Patience and perseverence are also the qualities required in the management of the deaf child. A decision has to be made whether to keep the child at home or to send it to a special training school for a time at least.

Treatment is based on establishing and developing alternative means of communication with the child and the outside world so that normal development can be allowed to proceed. Gestures, lip reading, pantomime and hearing aids all have their place in treatment.

## MIDDLE EAR INFECTIONS

These are a common cause of deafness. Deafness is usually only temporary. Children between 3 and 8 years of age are most often affected.

The mother or the schoolteacher are usually the first to suspect trouble. Confirmation is best carried out by means of audiometry and based on a series of recordings.

The condition is managed by treating the causal infection. Attention must also be paid to enlarged adenoids that may obstruct the eustachian tubes.

## TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Otitis media | History of previous ear troubles; usually at 3–8 years of age; drum abnormal | Clinical | (1) Treat otitis media<br>(2) Hearing aid if permanent<br>(3) Adenoidectomy when indicated | Deafness may be permanent in chronic otitis media |
| Wax in meatus | Obvious wax in meatus | Clinical | Remove wax | |
| Congenital | Associated with maternal rubella, congenital syphilis Kernicterus, genetic causes Often no cause found | Clinical (with difficulty) | (1) Early diagnosis<br>(2) Refer to special unit | Good prognosis with suitable training |
| Meningitis | Damage to auditory nerve following tuberculous, pneumococcal or meningococcal meningitis | Clinical | (1) Early diagnosis<br>(2) Refer to special unit | Prognosis depends on presence of other damage |
| Encephalitis | Associated with mumps and common fevers | Clinical | (1) Early diagnosis<br>(2) Refer to special unit | Prognosis depends on presence of other damage |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Trauma | Fractures at base of skull cause damage to auditory nerve | (1) Clinical<br>(2) Skull radiograph | (1) Suspect in cases of fractured skull base<br>(2) Early diagnosis<br>(3) If unilateral no special therapy needed | |
| Streptomycin | Chemical damage to auditory nerve and inner ear | Clinical | Check hearing in all children in whom streptomycin is used | Permanent damage if it occurs |

# DIARRHOEA

The frequency with which the bowels are open is very variable and the importance of diarrhoea lies more in an alteration in the character of the stools which become more fluid as well as more frequent.

## CAUSES

### ACUTE DIARRHOEA

*Infancy*

*Physiological*—It is quite normal for a healthy and thriving infant to pass 6–7 normal stools daily and this rate may vary at times and suggest 'diarrhoea'.

*Dietetic*—Underfeeding may cause 'hunger stools'—small, hard, green, mucus-covered stools passed frequently. 'Dietetic diarrhoea' due to wrong foods is rare and should not be used to explain uncertain situations. Likewise it is doubtful whether 'overfeeding' is often the cause of diarrhoea.

*Infection*—The most frequent type of acute diarrhoea is that due to mild gastro-intestinal infections associated with vomiting that come and are gone within 48 hours. No specific cause is usually found but many are probably virus infections. Other infective causes are: (1) epidemic diarrhoea (gastro-enteritis or summer diarrhoea), which may be due to viral or bacterial causes and is a serious condition in infancy; (2) staphylococcal entero-colitis, which is a 'hospital infection' and usually occurs in association with antibiotic therapy; (3) dysentery.

*Parental infections*—Diarrhoea associated with such other infections as pneumonia, otitis media and pyelitis.

*Local disease*—Intussusception and acute appendicitis.

*Childhood*

*Dietetic*—Said to occur after eating green apples, etc., but this is a risky diagnosis when abdominal pain and vomiting are also present.

*Infections*—Acute diarrhoea and vomiting of sudden onset lasting 48 hours is a frequent benign endemic and epidemic condition. It is probably a virus infection. The main infections are: (1) epidemic winter vomiting, which is a more specific entity often associated with diarrhoea; (2) food poisoning; (3) dysentery; (4) salmonella

infections (typhoid and paratyphoid); (5) staphylococcal enterocolitis.

*Parenteral infections*—Parenteral infections in chest, ears and urinary tract may be associated with diarrhoea.

*Local abdominal disease*—This may be acute appendicitis or ulcerative colitis (rare in children).

*Drugs*—The over-use of laxatives, especially senna and cascara; giving broad spectrum antibiotics, very frequently leads to diarrhoea.

### CHRONIC DIARRHOEA

*Physiological*—There is no set number of times that the bowels are open daily. 'Normal' may be up to 6 times or more.

*Steatorrhoea*—Excess of fat in the stools leads to frequent passage of bulky, pale and offensive stools. This is a feature of coeliac disease, and of fibrocystic disease of the pancreas.

*Nervous diarrhoea*—This is a real condition. It is associated with emotional upsets.

*Spurious diarrhoea*—This occurs in faecal impaction associated with constipation and in lesions of the central nervous system causing incompetence of the anal sphincter.

*Rare*

*Tuberculosis*—Tuberculosis of the intestines is now a rare condition.

*Ulcerative colitis*—This may occur as an unexplained mucoid diarrhoea which eventually becomes purulent and bloody.

*Amoebic dysentery*—Amoebic dysentery is rare in Great Britain, but can occur in immigrants from the tropics.

*New growths of large gut*—These are very rare in childhood but should be considered in obscure cases.

## PATHOLOGY

The effects produced by diarrhoea depend on its duration and severity; on the consistency of the stools (the more fluid the stools the more the disturbance because of loss of body fluids); on the age of the child (the disturbances will be much more severe in infants than in older children); and on the previous general health and on the social circumstances (the better these are the better the outlook).

*Biochemical Disturbances*

The functional disturbances in severe and continuing diarrhoea result from interference with the body's fluid and electrolytes.

These biochemical upsets may occur rapidly and catastrophically in young infants and require early diagnosis and urgent correction.

*Dehydration*—Loss of water in the stools causes dehydration. This is made worse if there is associated vomiting because the loss cannot be made good by drinking and more salts are lost in the vomit. Dehydration leads to decreased urine production and oliguria.

*Acidosis*—In addition to loss of water, acid-base balance is disturbed because of loss of base in the stools. This leads to acidosis. Acidosis is made worse by the impaired renal function associated with the dehydration. This low urine output leads to further retention of acid metabolites and failure to conserve base, and an increase in acidosis results. Renal impairment also leads to retention of urea with extrarenal uraemia. Potassium is lost from the intracellular fluid space for so long as acidosis exists and a potassium deficiency therefore results.

*Blood and urine examinations*—Correction of these acid-base and water derangements is an essential part of treatment. So that these can be assessed we need to know the amount of urine passed daily, and whether chlorides are present in the urine (chlorides are absent in dehydration). Blood examinations are necessary to estimate the levels of sodium, chloride and potassium. The alkali reserve of the plasma (plasma-bicarbonate) is lowered in acidosis. Elevation of the non-protein nitrogen indicates functional renal impairment. Haematocrit values and red blood cell counts serve as guides to the degrees of haemoconcentration or anaemia.

## DIAGNOSIS

### *History and Examination*

The history is of major importance. A description of what the mother means by the 'diarrhoea' is essential. Are the stools normal in consistency? Are they fluid? Are they hard? How often are they passed? A decision has to be made as to whether one is dealing with a normal physiological condition, an acute episode or a chronic disorder.

The general condition of the child must be assessed and is of more importance than the local digestive upset. Is the child apparently well or miserable and obviously unwell? Does he look dehydrated?

### *Associated Features*

Associated features to be considered are whether abdominal pain

is present, whether vomiting has accompanied the diarrhoea and whether there is any other primary condition present such as chest and ear infections or urinary upsets.

Questions relating to other members of the family will give a guide on the presence of an epidemic condition or possible food poisoning.

### BLOODY DIARRHOEA

Loose stools with blood-stained mucus raise different differential diagnostic problems and the following should be considered:

*Intussusception*—Blood and mucus passed but no real diarrhoea. Characteristic features are bouts of pain, collapse and the abdominal tumour.

*Henoch–Schönlein purpura*—Abdominal and joint pains followed by urticarial rash and blood and mucus per rectum.

*Rectal polyp*—Blood passed intermittently, usually with defaecation of normal stools.

*Ulcerative colitis*—Very rare in children. Pus, blood and mucus with fluid stools.

*Bacterial dysentery*—Acute diarrhoea with fever and fluid stools with bloodstained mucus.

*Amoebic dysentery*—Rare in Great Britain. Chronic diarrhoea, stools characteristically of 'anchovy sauce', that is altered blood in a fluid stool.

*Typhoid and paratyphoid infections*—These often present as cases of undiagnosed pyrexias. Diarrhoea may be insignificant with some blood and mucus in the stools.

## SIGNIFICANCE

Infants and young children with diarrhoea are very liable to severe general disturbances because of the rapidity with which dehydration and acidosis occur. Diarrhoea is therefore of great significance in these young children. The patients must always be under close supervision.

The dangers are greatest in infancy. Special care must be taken when the diarrhoea persists for longer than 24 hours, when the stools are very watery in consistency, when the social conditions are poor and when the mother is a 'non-coper'. The severity of the condition can be assessed clinically by dryness of the tongue and mucous membranes, by a sunken anterior fontanelle, by reduced tissue

turgor, by obvious loss of weight, by fever and by oliguria. Some or all of these factors indicate a potentially dangerous situation.

A decision has to be made whether the child should be admitted to hospital and although there are risks attached to placing a sick infant in hospital, for instance risks of cross-infection, it is only in hospital that the necessary investigations and correction of dehydration and electrolyte disturbances can be carried out.

## SPECIFIC CONDITIONS

### DYSENTERY

Acute bacillary dysentery is not uncommon in children. It tends to occur in epidemics that are caused by food contamination. The outbreak usually starts in schoolchildren and is then spread to other members of the family. It can occur at any time of the year but it is now most prevalent in winter months. In Great Britain *Shigella sonnei* is causal organism in 90 per cent of cases and *Sh. flexneri* in the remainder. The severe infections caused by *Sh. shigae* are never seen.

*Clinical Features and Course*

The onset is sudden, with frequent passage of watery inoffensive stools mixed with blood and mucus.

There is a variable amount of systemic upset. Fever is usually present. Colicky abdominal pains precede the passage of stools. Vomiting may be present but follows the onset of diarrhoea. The natural course is for the condition to clear up within 3–5 days. The diagnosis is confirmed by recovering the organisms from the stools by a rectal swab.

### FOOD POISONING

*Bacterial Toxins*

This type of food poisoning is caused by contamination of the food, usually a milk or cream preparation, with *Staphylococcus aureus*. The organisms produce a toxin which acts on the intestines. The onset is very sudden, with watery diarrhoea starting within 2 hours of eating the food.

*Bacterial Infections*

Bacterial infections are caused by eating egg or meat dishes contaminated by *Salmonella typhimurium*. The onset here is generally delayed for 24–36 hours from the time of eating the food.

*Poisonous Berries*

The berries of such plants as deadly nightshade and hemlock will produce diarrhoea, vomiting and collapse.

### STAPHYLOCOCCAL ENTEROCOLITIS

Although rare this is a notable complication of treatment of infections (usually respiratory) with broad spectrum antibiotics. During, or after, therapy diarrhoea occurs followed by high fever, vomiting, dehydration and shock.

### INFECTIVE NON-SPECIFIC DIARRHOEA

This is the most common type of diarrhoea in children. It is generally mild and self-limited. Severe and fatal cases still occur in infancy, though very much less frequently than in the past.

*Aetiology*

A virus is probably the responsible organism. The condition can occur at any age, but is most serious in infancy because of the risk of a biochemical complication. Severe cases occur only in bottle-fed babies and in the lower social groups.

*Clinical Features*

Mild cases occur suddenly and insidiously, with increasingly frequent motions. Vomiting is always an associated feature. The stools are yellow, watery and offensive at first and soon become green. The general condition remains good and resolution occurs within a week.

In severe cases the onset is sudden, with very frequent diarrhoea and vomiting. The stools are very watery and passed every half-hour or so. Vomiting is also severe and frequent. The general condition is poor, with dehydration and collapse, and the course may be fulminating with an appreciable mortality.

### STEATORRHOEA

Steatorrhoea implies the presence of excessive quantities of undigested fat in the stools.

The stools are characteristic in appearance. They are very bulky, pale, greasy and frothy. The smell is extremely offensive. The consistency is semi-fluid like gruel or thin porage, but bouts occur when the stools become loose and watery, but still with the terribly offensive smell and pale colour.

*Aetiology*

The causes of steatorrhoea are various. They are all concerned with abnormalities of fat digestion and absorption.

*Failure of digestion*—Jaundice (lack of bile salts); fibrocystic disease of pancreas (lack of pancreatic enzymes); giardiasis (worms line the intestinal wall and interfere with digestion of fats).

*Failure of absorption*—Cellular (coeliac disease); mechanical (tuberculosis of small gut; tuberculosis of mesenteric glands; Hodgkin's disease of mesenteric glands).

*Effects*

The effects of steatorrhoea are not confined to errors of fat digestion. There is always evidence of other deficiencies, such as anaemias, avitaminoses and general malnutrition.

## COELIAC DISEASE

An uncommon (less than 1 per 3,000 children) condition, in which there is a disturbance of absorption of various nutrients leading to wasting, infantilism, passage of large fatty stools and swelling of the abdomen. A familial tendency is present but the chance is only about 1 in 8 of another sibling being affected.

*Aetiology*

The aetiology is uncertain, but it is a fact that gluten protein in the diet makes the condition worse, while removing this from the diet results in control of the disturbance.

*Age Incidence*

It is a condition of infancy, being rare under 6 months of age, maximal at 1–3 years of age and rare after 5 years.

*Clinical Features*

The condition presents in three forms:

(1) Rarely, there is a tendency to loose and bulky stools from birth leading on to the more usual type.

(2) Classically, the onset is insidious between 6 months and 2 years of age, with loss of appetite, failure to thrive, irritability and loose, frequent and bulky stools.

(3) In an appreciable proportion the condition begins more acutely with a persistent 'diarrhoea' following on an infection such as bronchitis.

The signs are those of nutritional deficiency. There is loss of weight, particularly evident in the subcutaneous fat. The child fails

to grow and presents a stunted appearance with wasting (especially of the buttocks) and a protuberant abdomen distended with gas. Anaemia is often present and is of the iron-deficiency type. Rarely it may be macrocytic because of failure to absorb vitamin $B_{12}$. The various forms of vitamin deficiencies such as tetany and rickets (vitamin D and calcium) and scurvy (vitamin C) are now rare because of more effective treatment.

Stools are pale, bulky, loose and foul smelling. They look like porridge but may be frothy and greasy depending on the degree of fermentation. They may be passed frequently or only once a day. In the early stages or during exacerbations they are watery and fluid loss may cause dehydration.

*Investigations*

The characteristic findings of most useful tests are the low rate of fat absorption; the 'clumping' that occurs following a barium meal; the flat vitamin A absorption test; and the flat glucose tolerance curve.

*Differential Diagnosis*

Differential diagnosis must be from fibrocystic disease of the pancreas. In this the onset is much earlier, pancreatic enzymes are absent from the duodenum, there is an excess of salt in the sweat and a chronic lung infection is present. Other causes of steatorrhoea have also to be considered, such as giardiasis, where the parasite *Giardia lamblia* interferes with absorption; tuberculosis of the mesenteric lymph glands, now rare; and Whipple's syndrome.

*Course*

The natural history of the untreated case is for the acute phase to last for a few weeks to be followed by the more chronic condition. Improvement tends to occur spontaneously after some months or years. The diarrhoea improves first, followed by general physical and mental recovery. With treatment by gluten-free diet there is striking general improvement within a couple of weeks which is maintained while the diet is adhered to. Sometimes treatment is necessary up to puberty but usually it can be stopped by the age of 10 years. It is rarely fatal (approximately 5 per cent) and death is usually the result of lack of cooperation in treatment.

*Management*

The whole management is based on excluding gluten from the diet.

## TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Acute* | | | | |
| *Infancy* | | | | |
| Physiological | Normal variations are wide and the baby may pass up to 6 stools daily | Clinical | Reassurance | |
| Dietetic | Underfeeding more often a cause than overfeeding | Clinical | Advice | |
| Infection | Bacterial or viral causes; severity varies; mild cases common; severe cases rare but potentially fatal | Bacteriology | Severe cases must be treated in hospital | Mild, clear up *within* 48 *hrs.*; severe, are seriously ill |
| Parental infection | Associated with respiratory infections, pyelitis and other infections | Clinical | Treatment of primary cause | |
| Local disease | May be associated with intussusception and peritonitis | Clinical | Treatment of primary cause | |
| *Childhood* | | | | |
| Dietetic | Associated with dietetic indiscretions such as 'green apples' | Clinical | Symptomatic | |
| Infections | Bacterial or viral; food poisoning a frequent type | Bacteriology | Antibiotics if indicated | |
| Parenteral infections | Diarrhoea occurs with ear, chest, and urinary infections | Clinical | Antibiotics if indicated | |
| Local disease | Intussusception, appendicitis and ulcerative colitis | Clinical | Treatment of primary disease | |
| Drugs | Over-use of laxatives and with broad spectrum antibiotics | Clinical | Advice | |
| *Chronic* | | | | |
| Physiological | 'Normal' may be 6 or more stools daily | Clinical | Reassurance | |
| Steatorrhoea | Fatty diarrhoea associated with failure of digestion and absorption, for example coeliac disease, sprue and fibrocystic disease of pancreas | Special investigations | Special management depending on cause | Prognosis depends on cause |
| Nervous | Related to emotional upsets | Clinical | Advice | |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Spurious | With severe constipation and faecal impaction | Clinical | Treat cause of constipation | |
| Ulcerative colitis | Rare—persistent mucopurulent bloody diarrhoea | Sigmoidoscopy and barium enema | Special therapy | Poor outlook |
| Tuberculosis | Rare—Very sick child | Tuberculin test positive | Antibiotics | Fair prognosis |
| Amoebic dysentery | Rare—Occurs in children who have been in 'amoebic areas' | Stool examination for cysts | Specific therapy | Good outlook |

# EAR DISCHARGE

Although less frequent than in pre-antibiotic times, discharge from the ears is still a common problem in general and hospital practice.

## CAUSES

*Acute otitis media*—This is the most frequent cause of mucopurulent discharge. (See chapter on Earache.)

*Chronic otitis media*—This may follow acute infections but often it starts *de novo*.

*Otitis externa*—Otitis externa is an inflammation of the skin lining the external auditory meatus. The discharge consists of exudation from the affected skin.

*Boil in meatus*

*Foreign body in meatus*

## PATHOLOGY

Aural discharge is the result of disorders of the mucosa lining the middle ear or of the skin lining the outer ear. Associated with the discharge there is always some degree of deafness and hearing should be tested so that progress can be assessed.

*Outer Ear*

In conditions affecting the outer ear the disturbance is either an infection of the skin as in boils, an inflammation as in the eczematous lesions of otitis externa, with frequent secondary infection with pyogenic organisms, or irritation from a foreign body. Discharge from outer ear causes is serious to begin with but becomes purulent if secondarily infected.

*Middle Ear*

Otitis media is initially an infection of the mucous membrane lining the middle ear. Organisms gain access through the eustachian tube. This results in pain, deafness and discharge from the ear. Complete resolution is usual within a short time, but in a small

minority infection may persist with recurring episodes. This may be because of the virulence of the initial infection, because of spread to the underlying bone of the middle ear causing an osteitis or because of a persistent focus of infection in the nasopharynx with obstruction of the eustachian tubes by enlarged adenoids.

Acute and chronic otitis media are conditions with a different pathology, course and prognosis. Where the discharge persists for more than two weeks the condition must be regarded as 'chronic' and potentially progressive and dangerous.

## DIAGNOSIS

The correct diagnosis depends on a careful examination of the ear with an auriscope, an adequate history and on the mode of onset and the course.

It may seem simple to diagnose the presence of an intrameatal foreign body. So it is if the child is seen soon after the foreign body has entered the ear, but if forgotten until a few weeks or even months have passed the case will present as one of a purulent discharge from the ear with an infected mass of debris deep in the meatus. Only careful and gentle examination will reveal the true cause.

Differentiation of otitis externa from otitis media is far from easy. In both situations there is discharge with pain, deafness and fever. In the early stages the discharge in the external meatal condition is serous, whereas in otitis media it is mucoid or mucopurulent. Pain is produced in the former when the ear is moved. Examination of the meatus in otitis externa reveals a red weeping surface, while in otitis media the skin of the meatus is usually not primarily affected. In acute otitis media pulsatile discharge can be seen coming through the drum. In otitis externa the drum is intact.

The site and appearance of the perforation are of value in differentiating the two main types of chronic otitis media. Perforations with fairly clear-cut edges and sited in the lower and anterior part that are central and have some drum between it and the wall suggest an infection in the lower part of the tympanum from the nasopharynx. When the discharge is offensive and the perforation peripheral and in the upper and posterior part of the membrane the seat of the infection is almost certain to be in the attic and antrum of the middle ear with involvement of bone. This type is a serious and potentially dangerous condition.

## EAR DISCHARGE

### SIGNIFICANCE

Discharge is significant of some middle or outer ear disease, but before this is accepted it must be ascertained that there is actual discharge from the ear.

*Is there Discharge?*

Sometimes there is confusion in the parent's mind between normal wax and pathological discharge. Examination will also confirm whether discharge is coming from the middle or outer ear.

*What is the Cause?*

The chief decision to be made is in fact whether the discharge is coming from the middle ear or from some disorder of the outer ear.

The meatal skin must be inspected after the discharge has been wiped away with swabs. In middle ear conditions this skin will be normal, unless, as sometimes happens, there is a secondary otitis externa from irritating discharge. In otitis externa the meatal skin will be reddened, weeping and covered by scales or debris. The drum is intact in otitis externa and reddened with a pulsatile discharge and perforation in otitis media.

*What Should be Done?*

Local therapy is necessary in otitis externa and antibiotics in acute otitis media and a consultant's opinion in chronic conditions.

### SPECIFIC CONDITIONS

#### ACUTE OTITIS MEDIA

Seventy-five per cent of all acute middle ear infections occur in children under 10 years of age. Between 20 and 40 per cent of all children suffer one or more attacks in their childhood years.

Although there has been a decline in the severity of the condition, there does not seem to have been any reduction in the incidence of acute otitis media in the past 30 years.

The age incidence is similar to that found in upper respiratory infections. A peak period of susceptibility is between 3 and 8 years of age. There are differences in the incidence at various social levels; the severity is greater in the lower social classes.

*Clinical Features*

Discharge from the ear occurs in about a third of cases of acute otitis media. The course of the acute attack when discharge is present

is for the condition to settle within 14–21 days. Recurrences are frequent, occurring in no fewer than two-thirds of children. There is a definite tendency for the children to outgrow the attacks between 7 and 8 years of age.

*Complications*

Complications now are fortunately very rare. Mastoiditis has occurred in less than 1 per 1,000 attacks in reported series. Follow-up studies have shown some residual defects in between 1 and 5 per cent of children.

*Management*

Management depends on judicious use of antibiotics. Where recurrent attacks occur consideration must be given to the presence of any trouble in the nasopharynx, such as enlarged adenoids.

## CHRONIC OTITIS MEDIA

New cases of chronic middle ear disease are becoming rare, but are by no means unknown in children. In spite of apparently adequate therapy with antibiotics and other measures a small proportion will develop a chronic infection. This happens much more often in lower than upper social groups.

Any case where the discharge persists for longer than 4 weeks must be regarded as 'chronic' and steps taken to discover possible underlying causes. Chronic infection in the middle ear is usually maintained by a focus of infection either in the nasopharynx with enlarged and infected adenoids or in attic and antrum of the middle ear with an osteitis that may spread and involve the mastoid and possibly intracranial structures.

## OTITIS EXTERNA

Otitis externa is much less frequent than in adults. It is primarily an eczematous reaction of the skin lining the external auditory meatus. It may occur secondary to a discharging otitis media. Secondary infection with pyogenic bacteria or fungi frequently occurs. The condition tends to affect children with other seborrhoeic tendencies, and often there is a history of infantile eczema and wheezy chests.

*Clinical Features and Management*

The symptoms are clear serous discharge from the ear, pain and irritation (the older children complain of this). Examination of the

ear reveals a red, weeping and inflamed meatus with much debris. There is frequently an associated eczema of the skin surrounding the ear. The drum is intact but may be reddened because of the inflammatory reaction in the meatus. Recurrences are frequent.

Management is concerned with keeping the meatus clear of debris and discharge and applying bland and soothing local applications.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Acute otitis media | Sudden onset; fever; earache; mucopurulent discharge; normal meatal skin; red drum with pulsatile discharge | Clinical | (1) Antibiotics when indicated<br>(2) Adenoidectomy in recurrent cases | Good response; tendency to recur |
| Chronic otitis media | Persistent discharge; mucoid or mucopurulent; little general upset; perforation seen; normal meatal skin | Clinical | (1) Consultant's opinion<br>(2) Local toilet<br>(3) Adenoidectomy<br>(4) Mastoidectomy | Permanent damage may result; spread to mastoid and intracranial structures possible |
| Otitis externa | A skin disorder; pain; red, weeping, cracked meatal skin; drum normal; serous discharge; associated with seborrhoeic eczema elsewhere | Clinical | Local toilet and applications | Recurrent; no serious complications |
| Boil in meatus | Localized redness and swelling in meatus; painful and tender | Clinical | Antibiotics when indicated | |
| Foreign body in meatus | Discharge and deafness; history often not available; foreign body may be seen, or an infected granulating mass | Clinical | Removal of foreign body | |

# EARACHE

Earache is a frequent presenting 'symptom' in children from the age of 2 years upwards. Infants are unable to 'complain' of earache and the condition is manifest by more general disturbances.

## CAUSES

### OUTER EAR AND PINNA

*Boils*—These are rare under 3 years of age.

*Otitis externa*—This condition is uncommon in young children. (See chapter on Ear Discharge.)

*Trauma*—Trauma follows insertion of sticks or other objects.

*Foreign bodies*—These may be such objects as impacted beads, stones, etc.

*Wax*—Wax only causes pain if hard, dry and impacted.

### MIDDLE EAR

*Acute otitis media*—This is the most frequent cause of earache in children.

### REFERRED

*Throat and dental infections*—Such infections may present as 'earache'.

*Cervical lymphadenitis*—This may cause 'earache' by virtue of the child's inability to localize the pain.

*Mumps*—Mumps may also cause 'earache' through inability to localize the pain.

## PATHOLOGY

Earache is generally caused by involvement of pain-sensitive structures in the meatus and middle ear by inflammatory or traumatic conditions.

Acute otitis media is the most frequent cause of earache in children. Infection reaches the middle ear from the nasopharynx along the eustachian tube. Inflammation is associated with swelling of the

lining mucosa and accumulation of muco-pus in the cavity of the middle ear. These cause a rise of pressure and pain.

The external meatus is a sensitive structure and inflammation, as in boils and otitis externa, or trauma from injuries, hard wax or foreign bodies, may cause severe pain.

## DIAGNOSIS

The history will often only be presumptive and suggestive. The mother may herself think that her child has earache or there may be only rather vague features of a general disturbance with associated fever, irritability, catarrh, cough and vomiting.

The child should be examined generally in addition to local inspection of the ears. A routine should be followed to include the fauces, chest, abdomen, head, neck and limbs.

The pinna and meatus should be inspected in a good light. An electrical auriscope is generally used to inspect the drum. (Wax should be removed where present so that the drum can be seen.)

## SIGNIFICANCE

*Is Earache Present?*

This is often difficult to decide. Young children do not complain of 'earache'. They become irritable and cry, and have high fever, vomiting and diarrhoea.

The only way to determine whether there are any abnormal conditions in the ears is to examine them routinely in all sick children.

*What are its Causes?*

The cause of earache in any child can only be determined by careful examination. It is often difficult to decide whether the lesion is in the external or middle ear. The appearances of the skin of the meatus and the drum are the important features. In otitis externa there is redness and swelling of the skin with discharge from its surface. In acute otitis media the skin of the meatus is normal and the drum is abnormally red or perforated or both.

*What Should be Done?*

The diagnosis having been made a decision has to be taken as to what should be done. In inflammatory conditions the question is whether to use antibiotics or whether to observe the situation being

prepared to use them if necessary. Other causes such as impacted wax and foreign bodies require removal.

## SPECIFIC CONDITIONS

### BOILS

Boils (or furuncles) are a not uncommon cause of quite severe earache. They occur most frequently in older children, between 10 and 15 years, and are caused by superficial infection of the skin with *Staphylococcus aureus*. On examination the external meatus is almost occluded by red swelling of the walls of the meatus.

The most prominent feature is the extreme tenderness and the diagnosis can be made as the child screams when the auriscope is placed on the meatus.

### OTITIS EXTERNA

This is essentially an acute generalized inflammatory reaction of the external meatal skin. It may occur secondary to a discharging otitis media or as a primary condition with a normal drum and middle ear.

Clinically the main features are pain, irritation (older children complain of this), profuse thin aural discharge and swelling and crusting of the meatus. There is frequently an associated eczema of the skin surrounding the ear.

There is often a history of infantile eczema and wheezy chests in these children.

### TRAUMA

Most young children at some time play with their ears and put pencils, sticks and other objects into their ears. Abrasions and lacerations of the sensitive meatal lining are very painful and secondary infection is a usual complication.

### FOREIGN BODIES

Beads, pebbles, grass, flowers and other small things may be put into the ears and remain unnoticed until pain, deafness or secondary infection attract attention.

### WAX

Although less frequent than in adults, wax is often seen in children's ears. Painless as a rule, wax may cause discomfort when dry, when it then causes external meatal soreness.

## ACUTE OTITIS MEDIA

This is the condition *par excellence* responsible for earache in children.

*Incidence*

It is probable that almost half the population suffer one or more attacks of acute otitis media during their childhood years. The severity varies from minor and transient earaches to severe and continuing trouble with discharge and deafness. The incidence varies with social class (more frequent and more severe in lower social groups) and geography (more frequent and more severe in northern parts of Great Britain) (see page 93).

*Aetiology*

The bacterial infection is caused most frequently by staphylococci, pneumococci and haemolytic streptococci. These organisms reach the middle ear through the eustachian tube from the nasopharynx.

*Epidemiology*

More than 90 per cent of cases of acute otitis media occur in children. The usual age is between 3 and 8 years. It is a winter condition closely associated with the common respiratory infections of childhood.

*Associated Symptoms and Signs*

Pain, namely earache, is the presenting symptom. This earache is severe and is worse at night. Examination will show a red drum, sometimes with blisters on its surface. In about a third of cases of otitis media discharge of muco-pus occurs from the meatus.

Deafness is the other notable feature and care has to be taken in following up these children until normal hearing is restored.

*Course*

Reference has been made to the peak incidence between 3 and 8 years of age. Even in those children with a history of recurrent attacks after the age of 8 years the attacks become less and less frequent. Recurrent attacks are common. The natural tendency is for a spontaneous cessation of attacks after this age of 8 years.

*Management*

The children must be followed up until the appearance of the drum returns to normal and normal hearing is restored.

Decisions that have to be made are:

(1) Whether to use antibiotics or to wait and see for a day or two.

(2) Whether adenoids and tonsils should be removed.

(3) When to refer to a consultant in cases of persistent discharge and deafness and fever.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Acute otitis media | Sudden onset; pain, fever and earache; discharge may occur; red drum | Clinical | (1) Symptomatic (2) Antibiotics | Many settle on non-specific measures |
| Boils | Pain; tenderness, red swelling in meatus | Clinical | (1) Symptomatic (2) Antibiotics | |
| Otitis externa | Pain and discharge from ear; red weeping skin of external meatus; drum normal | Clinical | Local applications | Tendency to recur |
| Wax | Dry and impacted wax | Clinical | Remove by syringing | |
| Foreign body | Impacted, causing pressure on drum or ulceration of meatal wall | Clinical | Removal | |
| Trauma | Damage to meatal walls | Clinical | Local cleansing | |
| Throat and dental infections | Referred pain associated with acute sore throat or infection | Clinical | Treatment of primary infection | |
| Cervical adenitis | Failure to localize site of pain which is referred to region of ears; clinical evidence of primary condition | Clinical | Treatment of primary condition | |
| Mumps | Failure to localize site of pain which is referred to region of ears; clinical evidence of primary condition | Clinical | Treatment of primary condition | |

# EATING PROBLEMS

Two common problems often face the doctor—the child who 'won't eat' and the overeating obese child.

## CAUSES

'*Not Eating*'

*Physiological*—Parents tend to overestimate their children's capacities.

*Negativism*—A normal but difficult phase during which the child refuses to eat what, and as much as, his mother wishes.

*Psychotic* '*Anorexia nervosa*' (*rare*)—Occurs principally in adolescent girls.

*Organic* (*rare*)—Chronic infections and new growths.

*Overeating*

*Physiological*—A 'good healthy appetite'.

*Psychological*—In some deprived children overeating compensates for a lack of love and attention.

*Iatrogenic*—Treatment with steroids leads to a marked gain in weight.

*Central nervous system* (*rare*)—Mental defect, frontal tumours, hypothalamic tumours.

*Endocrine* (*rare*)—Pituitary, adrenal and sex gland disorders.

## PATHOLOGY

Appetite is a pleasurable sensation that may be affected by emotional and physical states. It also varies with constitutional and familial factors. Some babies and infants, as well as children and adults, have a larger capacity and inclination for food than others.

Hunger, on the other hand, is an unpleasant sensation. It is a painful experience associated with strong peristaltic contractions of the stomach. It is certainly experienced by infants as well as older children.

Normally, hunger determines when a person eats, appetite what he eats and satiety how much he eats.

## DIAGNOSIS

Appetite is such a sensitive mechanism that it is easily influenced by a variety of causes.

By far the most frequent causes of abnormal appetites are emotional influences affecting the child or merely physiological variations of the normal.

'*Not Eating*'

The child who won't eat what his parents expect him to is most likely to be suffering from an emotional disturbance. However, this must be differentiated from possible organic causes which, although very much rarer, must be considered.

These organic causes include acute and chronic infections, particularly those of the respiratory and renal tracts. Tuberculosis is now becoming increasingly rare but must be considered in any general disturbance in childhood. Metabolic upsets in diabetes mellitus and renal failure are other rare causes of anorexia. New growths, among which leukaemias are a prominent group, are also rare and soon present symptoms and signs pointing to their diagnosis.

*Overeating*

Most of the 'overeaters' also belong to the simple and relatively benign physiological or emotional groups and only rarely do the more exotic causes such as endocrine and neurological lesions have to be considered.

## SPECIFIC TYPES

### NON-EATERS

Refusal of food by children seems to be peculiar to modern Western civilization. The syndrome is most prevalent in middle-class society where mothers are now forced to try to do more and more for their children on their own initiative. The mothers aim to achieve perfection but this leads to considerable lack of self-confidence in the child.

One of the main concerns of a young mother is that of feeding her young and endeavouring to make sure that they are receiving sufficient food. She tends to assess the quantities required from books and periodicals and from her own estimates of what they eat and of what her friends say that their own children are eating.

This concern with feeding makes every meal a trial and a challenge. The mother's stress and tension tend to be transmitted to her child and a state of anxiety develops. The child also becomes tense and irritable and reacts by refusing food. Cajoling, threats and fussing by the parents only aggravate the situation and a vicious circle develops.

The condition is most frequent in the second and third year of life. The mother brings the child for advice. Usually, in spite of the story of non-eating, the child is well covered, not underweight, in apparent good health, and obviously is getting enough food. Reassurance, explanation and advice are required, usually repeatedly, and sometimes reinforced by a second opinion.

A second period at which 'non-eating' is a problem is in the first couple of years at school. Here the cause also is partly emotional but is largely due to the repeated infections to which the child is subject producing a general state of debility.

## 'OVEREATERS'

Overeating is the most frequent cause of obesity, but constitutional and emotional factors are also of importance.

The condition tends to present in early school life between the ages of 6 and 9 years. The mother brings the child because of the taunts to which he is subject at school.

These children tend to be taller and generally larger than average. The fat is distributed principally over the breasts, hips and abdomen. The genitals are normal, but may be hidden in the fat.

Even if no treatment is given there is a tendency for these children to outgrow their obesity with adolescence.

# FAILURE TO THRIVE

Regular and progressive gain in weight and the passing of the milestones of development at the accepted times are features of the normal. Any persistent departure from this pattern requires a consideration of possible troubles.

## CAUSES

*Inadequate Food Intake*

Inadequate food intake may result from:

(1) Economic or social privations.

(2) Anorexia secondary to psychological or organic conditions.

(3) Mechanical feeding difficulties:

(a) Cleft palate and hare lip, congenital short oesophagus and others.

(b) Dyspnoea due to any cause.

(c) Mental or physical retardation for various reasons, which causes infants to feed poorly.

(4) Underfeeding due to fear of overfeeding and over-rigid adherence to feeding charts.

*Failure to Assimilate Food*

Failure to assimilate food may result from:

(1) Inadequate digestion in fibrocystic disease of the pancreas, which causes an early failure to gain weight.

(2) Inadequate absorption which will occur in the coeliac disease syndrome, though failure to thrive is not an early symptom. Vomiting and perhaps diarrhoea may occur first, followed by a period of irregular weight gain. Loss of weight becomes prominent in the more advanced stages.

*Loss of Food Substance*

Actual loss of food will occur in all conditions associated with vomiting and diarrhoea that last longer than a few days.

*Failure of Utilization and Metabolism*

A group that includes a wide variety of different conditions, as follows.

(1) Recurrent or persistent infections. These may be:

(a) Respiratory infections, for example bronchiectasis and tuberculosis.

(b) Urinary infections, especially when associated with an underlying congenital defect that predisposes to recurrences.

(c) Intestinal parasites, such as tapeworms.

(2) Malignancy, which should be considered in a child with a history of weight loss or unexplained listlessness. Leukaemia, Hodgkin's disease and bone tumours should be borne in mind.

(3) Congenital heart anomalies, in which failure to gain weight may be an early feature and is an indication of inadequate oxygenation of the blood.

(4) Renal insufficiency, which is an important cause of failure to grow and gain weight. Renal acidosis in infants is associated with anorexia, vomiting, constipation and failure to gain weight. Symptoms begin between 3 and 6 months of age. Blood chemistry shows an acidosis with high levels of chlorides but the urine is alkaline or neutral in reaction. Idiopathic hypercalcaemia is clinically indistinguishable from infantile renal acidosis but here there is a high serum calcium.

(5) Hepatic failure from cirrhosis of various types, which will lead to failure to thrive.

(6) Endocrine disorders such as hyperthyroidism and hypothyroidism, diabetes mellitus and hypoadrenalism, which all lead to loss of weight and failure to progress.

(7) Metabolic and storage disorders, as follows:

(a) Glycogen storage disease of the liver (von Gierke's disease), a rare condition with marked liver enlargement and almost constant ketosis. It is inherited as Mendelian recessive and is a defect of the enzyme system controlling glycogen and dextrose metabolism. In mild cases the condition escapes notice until the second year when dwarfing is noted together with a large abdomen. In severe cases symptoms of hypoglycaemia are a feature with vomiting, drowsiness, twitching or convulsions. Glycogen storage disease may also affect the heart, producing heart failure in the first few months of life.

(b) Galactosaemia, a rare familial disorder of infants with jaundice; failure to gain weight is an early feature with bouts of diarrhoea and vomiting. The liver becomes enlarged and there are also mental deficiency and often lamellar cataracts if the condition remains untreated. Galactose and albumin are found in the urine. The primary defect is a deficiency of a specific enzyme

present in erythrocytes which interferes with the metabolism of galactose which is derived from lactose in the milk. (See also chapter on Jaundice.)

(c) Cystinosis, in which failure to thrive is an early feature. Cystine storage disease (Lignac–Fanconi syndrome) is a very rare familial disease with thirst, polyuria, arrested growth and rickets. Cystine crystals are deposited in many organs, especially the bone marrow, liver, spleen and in the cornea (where they can be seen by a slit-lamp). The urine contains amino acids and glucose.

(d) Gaucher's disease, which in infants is characterized by physical and mental retardation with enlargement of the spleen and liver and with neurological signs such as alterations in muscle tone, dysphagia, laryngeal spasm and trismus. The cause is a familial defect in an enzyme concerned with cerebroside metabolism.

(e) Niemann–Pick disease, a very rare condition due to disturbance of phosphatide metabolism and is manifested by emaciation, enlargement of spleen and liver and mental retardation.

(f) Severe cerebral damage from trauma and cerebral palsy or other causes, which sometimes interferes with growth.

(g) Unknown cause, a final group of those who fail to thrive in whom no cause can be found.

## PATHOLOGY

The detailed pathology of the various conditions leading to failure of a child to thrive depends on the individual disorder, but in general terms there are three possible stages at which troubles may occur.

(1) Food intake and loss. Obviously, unless there is an adequate qualitative as well as quantitative intake of food there will be a deficiency in raw materials from which growth and development may take place. Failure in this respect may result from avoidable or unavoidable deprivation of food, from mechanical defects in the digestive passages or from a loss of food by vomiting, or from too rapid passage through the intestines.

(2) Digestion and absorption. Once the food has passed through the stomach it has to be digested and absorbed in the intestines. Failure may occur because of defects in the digestive juices as in fibrocystic disease and jaundice or because of troubles with actual absorption of the digested food through the intestinal walls as in coeliac disease, mesenteric tuberculosis and certain other mechanical defects.

(3) Metabolism. Digested food stuffs that have been absorbed into the circulation have then to be metabolized if they are to benefit the child. A large number of rare metabolic defects have been defined in recent years. They are concerned with the deficiencies of intracellular enzyme systems. These deficiencies may lead to failure to thrive in addition to many other possibilities such as mental retardation and accumulation of non-metabolized substances in the liver, spleen and other parts of the reticulo-endothelial system.

## DIAGNOSIS

The diagnostic approach to the child who fails to thrive should be systematic.

First, a decision has to be made as to whether the failure to thrive is the result of physiological 'slowness' of development or alternatively is the consequence of some underlying pathological cause.

Second, having decided on the probability of an organic cause the mental exercise of going through the broad possible aetiological groups is a useful one.

Third, there comes the clinical exercise of assessing the mother's history together with the physical signs to lead one to a likely diagnosis which can then be confirmed by investigations, many of which are now highly specific.

## SIGNIFICANCE

The greatest gain in weight during foetal development occurs just before birth owing to deposition of fat in the last trimester. A physiological loss of weight due to loss of fluid occurs in the 3 or 4 days after birth. Usually the birth weight is regained by the tenth to the fourteenth day.

Weight gain during infancy is relatively rapid amounting to from 4 to 10 oz. a week. The rate of gain is slower in the second six months of life, being about 3–5 oz. a week.

During the first year of life infants should demonstrate a steady weight gain, for this is the best indicator of normal nutrition and health. Failure to do so calls for investigation.

As a general rule if an infant fails to gain on a diet that is apparently quantitatively and qualitatively correct, then there is something wrong with the infant or the feeding situation and not with the formula or type of milk. Repeated alterations of the formula or changing to other preparations does not help the situation but merely serves to confuse the issues.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Inadequate food intake* | | | | |
| (1) Economic and social deprivation | National and individual problem | Clinical | Adequate diet | Depends on development |
| (2) Secondary anorexia | Anorexia secondary to emotional or organic causes | Clinical | Treatment of causal condition | Depends on cause |
| (3) Mechanical difficulties | Cleft palate, congenital hiatus hernia | (1) Clinical<br>(2) Radiograph | Surgery when indicated | Good |
| (4) Underfeeding | Overanxious mother; baby cries a lot and fails to gain | Clinical | (1) Adequate diet<br>(2) Reassurance of mother | Good |
| *Failure to assimilate* | | | | |
| (1) Inadequate digestion | Fibrocystic disease of pancreas; non-digestion of fats and protein; steatorrhoea; chest infections; failure to gain weight and grow | (1) Clinical<br>(2) Faeces<br>(3) Sweat electrolytes<br>(4) Duodenal juice | Antibiotics and pancreatin | Poor |
| (2) Inadequate absorption | Coeliac syndrome; stunted growth, swollen abdomen, wasted buttocks, steatorrhoea | (1) Clinical<br>(2) Faeces<br>(3) Fat absorption poor<br>(4) Flat glucose tolerance test | Gluten-free diet | With treatment good outlook; without treatment death may occur |
| *Loss of food* | | | | |
| Vomiting and diarrhoea | Rapid loss of weight in acute cases due to infections | (1) Faeces<br>(2) Serum electrolytes | Treatment of infection and fluid disturbances | Good with treatment |
| *Failure of metabolism* | | | | |
| (1) Recurrent infections | Chronic and recurrent respiratory, renal and intestinal infections | (1) Clinical<br>(2) Urine<br>(3) Chest radiograph<br>(4) Faeces | Treatment of cause | Depends on course |
| (2) Malignancy | Must be considered in unexplained loss of weight and persistent listlessness | (1) Clinical<br>(2) Blood<br>(3) Biopsy | Depends on type of malignancy | Bad |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| (3) Congenital heart disease | Failure to grow is an early feature in cyanotic heart disease | (1) Clinical<br>(2) Special cardiological investigations | Depends on type | Depends on type |
| (4) Renal insufficiency | Rare—Renal acidosis with anorexia, vomiting, constipation and failure to gain weight; 3–6 months of age | (1) Blood: acidosis and high chlorides<br>(2) Urine: alkaline or neutral | Alkalis by mouth and measures to correct dehydration | Good with treatment |
| (5) Hepatic failure | Rare—Follows infective hepatitis or other causes of cirrhosis | (1) Clinical<br>(2) Liver function tests | Depends on cause | Depends on cause |
| (6) Endocrine disorders | Rare—Diabetes mellitus, thyroid disorders and hypoadrenalism | Depends on type | Depends on type | Depends on type |
| (7) Metabolic storage disorders | | | | |
| (a) Glycogen storage (von Gierke's disease) | Rare—Swollen abdomen, liver enlargement and ketosis | (1) Clinical<br>(2) Ketosis | Symptomatic treatment of hypoglycaemia | Poor |
| (b) Galactosaemia | Rare—Neonatal jaundice; liver enlarged; mental defect; lamellar cataracts; hepatic and renal failure | Urine contains reducing substance, albumin and amino acids | Lactose-free diet | With early diagnosis and treatment, fair outlook |
| (c) Cystinosis | Rare—Thirst, polyuria, rickets and arrested growth; cystine crystals seen in cornea with slit-lamp | (1) Urine: amino acids and glucose<br>(2) Corneal: cystine crystals | Symptomatic | Bad; death from renal failure |
| (d) Gaucher's disease | Rare—Error of cerebroside metabolism, liver and spleen enlarged; interference with muscle tone; mental and physical retardation | (1) Radiograph of bones shows osteoporosis (focal)<br>(2) Marrow biopsy shows Gaucher cells | Symptomatic | Poor, but may live for years |
| (e) Niemann–Pick disease | Rare—Error of phosphatide metabolism; progressive emaciation; liver and spleen enlarged | Bone marrow: 'Foam cells' present | Symptomatic | Fatal |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| (f) Cerebral damage | Rare—Severe cerebral damage from trauma, cerebral palsy and other causes; failure to thrive | Clinical | Symptomatic | Poor |
| (8) Unknown cause | No cause may be found in some instances | Clinical | Symptomatic | Uncertain |

# HEADACHE

'Headache' is not a very common symptom in children and should always be taken seriously.

## CAUSES

### GENERAL

*Infections*—Infections of all types can produce headaches.

*Emotional upsets*—Such upsets are much less often a cause than in adults but still a frequent one.

*Acute nephritis*—In acute nephritis, headache occurs as a result of the rise in blood pressure.

### LOCAL

*Extracranial*

*Myalgia*—Muscular lesions, such as those produced by trauma, may cause headache.

*Eyestrain*—Eyestrain is a possible cause of headache in older children.

*Spine*—Rarely lesions of the cervical spine (such as new growths and osteomyelitis) may cause occipital headache.

*Cranial*

*Acute otitis media*—This condition may lead to a complaint of headache. (See chapter on Earache.)

*Sinus headaches*—Sinus headaches are rare in children. When present they are paroxysmal and tend to occur regularly at about the same time each day.

*Lesions of the skull*—Such lesions are rare, for instance Hand-Schüller–Christian syndrome.

*Intracranial*

*Trauma*—For instance, chronic subdural haematoma.

*Infection*—For instance, meningitis, brain abscess, encephalitis.

*Vascular*—For instance, subdural haematoma, subarachnoid haemorrhage, migraine.

*Neoplasms*

## PATHOLOGY

The brain itself is insensitive to pain. The blood vessels with their sensory nerves are the sensitive structures. Dilation, stretching and traction of these tissues cause headache in infections, migraine and space-occupying lesions.

Headache from extracranial causes is the result of reflex involvement of the neck and eyes. When it does occur it is usually the result of some relatively minor disturbance; the symptom must always be taken seriously as due to a potentially serious cause.

## DIAGNOSIS AND SIGNIFICANCE

An accurate diagnosis of the cause of headache will depend on a careful clinical assessment of the symptoms and signs. A systematic approach is necessary.

*History*

The duration of the symptoms will help to differentiate acute and more dramatic conditions from more chronic, persistent and recurrent states.

The mode of onset may be sudden and dramatic as in subarachnoid haemorrhage, slowly progressive as in intracranial space-occupying lesions, periodic as in migraine and constant as in psychogenic headaches.

A history of trauma may be of help or merely a 'red herring'. The possibility of a subdural haematoma must be considered when trauma is mentioned, but on the other hand in normal and active children some traumatic experience can almost always be recalled.

The severity of the headache will help to distinguish serious causes such as meningitis and space-occupying lesions from extracranial general causes and migraine.

*Site of the Headache*

The site of the headache is of value. Unilateral headache is a classical feature of migraine, but in children and when the headache is fairly severe such localization is by no means always present. In general localization is not a striking feature in childhood, but certain points are worth noting.

Occipital headache is often associated with lesions in the cervical spine and muscles. A sudden and severe occipital headache occurs

in subarachnoid haemorrhage and pain here is also an early feature of poliomyelitis.

Frontal headache is often ascribed to eyestrain but it may be present in frontal sinusitis.

*Associated Symptoms*

Associated symptoms must be considered. Vomiting in association with headache is always of significance. It may be the result of either general or local causes.

Fever may be the result of localized infection such as meningitis or brain abscess but it is much more likely to be associated with a general disturbance or infection in which headache is an incidental symptom.

The presence of convulsions and loss of consciousness both suggest serious intracranial causes.

## SPECIFIC CAUSES

### MIGRAINE

Migraine is chiefly a condition of adults with a peak prevalence between 20 and 50 years of age. In adults it is three times as frequent in women as in men. Although uncommon, migraine does occur in childhood. It is very unusual before the age of 10 years. Boys and girls are equally affected.

*Clinical Features*

The main features of migraine are headache, vomiting and visual disturbances. In adults the first symptom is often a transient visual disturbance such as fortification spectra or hemianopia followed a short while later ($\frac{1}{2}$–1 hour) by a unilateral headache and vomiting.

In children the visual disturbances are rarely mentioned and the headache tends to be generalized rather than unilateral. Vomiting is a prominent symptom and may precede the headache and be repeated. Abdominal pain is often a part of the migraine syndrome in children.

Migraine is a recurrent condition. The frequency may vary from once a week to once a year. A positive family history, especially on the mother's side, is another characteristic.

The relationship between migraine and the periodic syndrome is a close one. Many believe that the periodic syndrome is in fact a migraine-equivalent. Children with the periodic syndrome often develop migraine later in life.

### INFECTION OF THE NERVOUS SYSTEM

Three types of infection of the nervous system that can cause headache are meningitis, encephalitis and poliomyelitis.

### MENINGITIS

Meningitis may be acute when the most frequent causal organisms are viruses, meningococci, pneumococci, *Haemophilus influenzae*, streptococci and staphylococci. Chronic meningitis may be the result of infection by tubercle bacilli and the spirochaete of syphilis.

*Clinical Features*

Meningitis is most often a disease of infancy. Clinically the features are those which result from irritation of the meninges and those that are associated with septicaemia.

The onset may be acute, with fever, rigors, headache (when the child is able to complain) and vomiting. Convulsions occur in infants. The child tends to be drowsy and delirious, photophobia is present and if untreated the child becomes comatose and death from exhaustion and toxaemia follows. In infants bulging of the anterior fontanelle is a sign of the raised intracranial pressure present in meningitis. On the other hand the onset may be less dramatic, with general malaise, fever, headache and vomiting.

Signs of meningeal irritation include neck stiffness and a positive Kernig's sign.

In meningococcal meningitis a macular and petechial rash is present on the trunk and buttocks.

*Diagnosis*

The diagnosis must always be confirmed by examination of the cerebrospinal fluid. The pressure of the cerebrospinal fluid is increased in meningitis. Normally cerebrospinal fluid is clear and colourless, in meningitis it ranges from hazy to frankly purulent. The causal organisms can usually be isolated from the cerebrospinal fluid. The number of cells is much increased, as is the protein content. The level of sugar in the fluid falls.

*Course*

Without treatment the majority of children with meningitis will die. With specific therapy the outlook is fair, but complications are not infrequent.

### ENCEPHALITIS

The term 'encephalitis' tends to be loosely applied to conditions in which there is acute and diffuse damage to the brain by viruses, bacteria, toxins or poisons. In some no cause can be found. The damage may take the form of direct injury to the nerve cells, demyelination, oedema, granuloma, thrombosis or haemorrhage.

Encephalitis is a rare condition. The most frequent type is that which complicates common infections such as measles, chicken-pox, mumps, rubella and vaccinia. The onset is usually 7–21 days from the start of the infection.

*Clinical Features and Prognosis*

The clinical features include fever, headache, signs of meningeal irritation, disturbances of consciousness (such as confusion, delirium, drowsiness, stupor and coma), convulsions and functional disturbances (such as paralysis, blindness, deafness, ataxia and involuntary movements). The abnormal signs are liable to frequent alterations.

There is no specific treatment for encephalitis and the outlook is not good. There is an appreciable mortality rate of 10–50 per cent and permanent complications are frequent.

### POLIOMYELITIS

The poliomyelitis virus is endemic in the community but severe paralytic attacks are fortunately rare.

*Paralytic Form*

The classical paralytic form is the least common type. The first phase is that of a generalized infection, with malaise, fever, aching limbs, sore throat and a diffuse headache that is often maximal in occipital region. In the second paralytic phase fever persists, there are signs of meningeal irritation with neck stiffness, a positive Kernig's sign, spasm of the spinal muscles causing an inability to kiss the knees and a positive 'tripod sign'. The paralysis, once it appears, is not necessarily maximal at the outset but may develop over a few days.

*Non-paralytic Form*

In the more common non-paralytic form headache and general malaise, fever and signs of meningeal irritation are prominent. It is this type that has to be differentiated from other causes of non-bacterial meningo-encephalitis.

### SPACE-OCCUPYING LESIONS

Three types of space-occupying lesions may be recognized—new growths, post-traumatic subdural haematoma and brain abscess.

The rise in intracranial pressure associated with these lesions causes the following symptoms and signs.

*Headache*—Headache is a prominent feature in the older children. It tends to be paroxysmal and worse on lying down, on awakening and first thing in the morning and on coughing, bending and straining.

*Vomiting*—This is almost always present. It may occur quite suddenly and apparently spontaneously.

*Papilloedema*—This is a late sign.

*Convulsions and mental changes*—Convulsions do not occur in all cases and mental changes are unusual and occur late in the course of these conditions.

Any suspicion of a space-occupying lesion must lead to careful investigations to determine the cause.

### TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| General | Infections of all types can cause headache<br>Emotional upsets are a frequent cause of vague headaches<br>Acute nephritis: headache results from hypertension and toxaemia | (1) Clinical<br>(2) Specific tests where indicated | Depends on primary condition | Depends on primary condition |
| Extra-cranial | Eyestrain is a possible cause of headache in older children<br>Disorders of cervical spine and cervical muscles may cause occipital headaches | (1) Clinical<br>(2) Radiograph of cervical spine | Depends on primary condition | Depends on primary condition |
| Cranial | Sinusitis may cause frontal headaches<br>Acute otitis media may cause the child to complain of vague headache<br>*Rare*—Skull lesions are rare causes of headache | (1) Clinical<br>(2) Radiograph of skull | Depends on primary condition | Depends on primary condition |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Intracranial | Trauma may lead to formation of subdural and extradural haematomas which act as space-occupying lesions and cause persistent headache and vomiting<br>Infections, for instance, meningitis, brain abscess and encephalitis, will all cause headaches<br>Migraine is a frequent cause of headaches in older children<br>New growths will lead to symptoms of raised intracranial pressure | Depends on primary condition | Depends on primary condition | Depends on primary condition |

# JAUNDICE

The characteristic yellow staining of the skin, mucous membranes, sclerae and plasma in jaundice is the result of an excess of bilirubin in the circulation.

Jaundice is no single or separate disease but rather the symptom of many possible disease processes.

## CAUSES

There are a number of ways of classifying the types of jaundice. None is absolutely satisfactory because there is often more than one cause responsible for the condition.

In childhood the most practical method is to consider the causes at three age periods—neonatal, infancy and in older children—and to subdivide the actual types into pre-hepatic, hepatic, and post-hepatic.

In pre-hepatic jaundice the cause is in the blood. The jaundice results from an excessive destruction of red blood cells by haemolysis.

In hepatic jaundice the pathology is in the liver cells, which are unable to carry out their functions of excreting bilirubin.

In post-hepatic jaundice the cause is in obstruction of the cystic or bile ducts.

### NEONATAL PERIOD

*Pre-hepatic Jaundice*

*Physiological*—This is an invariable feature in prematurity but may also occur in normally mature babies. It is really a mixture of pre-hepatic and hepatic causes. The immature liver is unable to deal with the red blood cells that are haemolysed. It occurs within 2–5 days of birth (never at birth) and persists for less than a week.

*Haemolytic disease of the newborn*—This results from incompatibility of blood groups in infant and mother.

*Hepatic Jaundice*

*Galactosaemia*—A rare familial disorder.

*Toxoplasmosis*—The infecting organism is a protozoon. Although actual infection is common, symptoms and signs are rare. The main

features are in the central nervous system with choroidoretinitis, hydrocephalus or microcephaly and fits. Jaundice, with a large liver and spleen, may be a sign in the neonatal period, together with enlargement of the lymph glands, oedema and skin rashes.

*Congenital syphilis*—This is now very rare but it may occur if routine blood tests in pregnancy are omitted. Jaundice associated with a large liver is a feature.

*Post-hepatic Jaundice*

*Congenital obliteration of the bile ducts*—This is rare. Progressively deepening jaundice is present from birth. Diagnosis is not usually made until about the third week because of the comparison with physiological jaundice. The disease is fatal unless surgical relief is possible.

### INFANCY

*Pre-hepatic Jaundice*

*Acholuric jaundice*—Acholuric jaundice, that is jaundice with no bile in the urine, results from a form of haemolytic anaemia. The cause is an abnormal fragility of the red blood cells, which are spherocytic. Excessive destruction of these blood cells occurs with the production of large amounts of bilirubin. The spleen is enlarged. Pigment gall-stones occur. The course is marked by periodic crises with worsening of anaemia and increase of jaundice. (See chapter on Anaemia.)

*Hepatic Jaundice*

*Infective hepatitis*—This condition is unusual under 3 years of age. There is a mixture of hepatic and obstruction (post-hepatic) factors.

*Post-hepatic Jaundice*

*Biliary duct obstruction*—This is rare in infancy.

*Choledochus cyst*—This also is rare, with no symptoms until 5–12 years of age. It only occurs in girls. There is congenital cyst formation and dilatation of common bile duct. Clinically the characteristics are intermittent attacks of jaundice with vague aching in upper abdomen. A cystic mass is felt in the right upper abdomen.

### OLDER CHILDREN

*Pre-hepatic Jaundice (rare)*

*Acholuric jaundice*—(See chapter on Anaemia.)

*Sickle-cell anaemia*—The sickle-shaped red blood cells are fragile

and easily destroyed, resulting in a haemolytic anaemia. (See chapter on Anaemia.)

*Malaria*—Jaundice results from the destruction of red cells by the parasites.

*Other types of haemolytic anaemia*

*Hepatic Jaundice*

*Infective hepatitis*—Acute liver infection caused by virus.

*Homologous serum jaundice*—This is also an acute viral infection of the liver, transmitted by contaminated syringes or needles used for injections or blood transfusions.

*Weil's disease*—This is a generalized infection caused by the spirochaete *Leptospira icterohaemorrhagiae* and is acquired by walking or bathing in water contaminated by infected rat's urine. It occurs most often in campers and Boy Scouts. The clinical manifestations are fever, limb pains, headache, red eyes, skin haemorrhages, albuminuria and oliguria and jaundice.

*Poisons and drugs*—Jaundice from these causes is rare in childhood but liver damage can result from chloroform, carbon tetrachloride and phosphorus poisoning.

*Post-hepatic Jaundice*

*Bile-duct obstruction*—This may be either extrinsic (caused by enlarged lymph glands, neoplastic deposits or cysts) or intrinsic (caused by gall-stones or roundworms).

## PATHOLOGY

Jaundice results from an excessive amount of bile pigments in the blood. The normal amount of bilirubin, the most important of these pigments, is 0·2–0·8 mg per 100 ml. Clinically the yellow colour of jaundice is first apparent when the level of bilirubin reaches 2 mg per 100 ml.

To understand the pathogenesis of jaundice the fate of the haemoglobin must be studied.

The haemoglobin of effete red blood cells is broken down in reticulo-endothelial cells, chiefly of the liver and the spleen. The 'haem' part is converted into bilirubin and this enters the circulation and passes to the liver. The 'pre-hepatic' bilirubin is chemically different from the 'post-hepatic' bilirubin that has passed through the liver.

In the liver the bilirubin becomes one of the constituents of bile which is collected into the intrahepatic biliary canaliculi and thence to the bile ducts. Stored in the gall-bladder it then passes along the common bile duct to the duodenum. In the intestines bilirubin is reduced to urobilinogen. Some of this is reabsorbed into the circulation and passes to the liver and kidneys where it is re-excreted in the urine, but most is excreted (as sterocobilin) in the faeces.

Pre-hepatic jaundice occurs when excessive numbers of red blood cells are destroyed. Bilirubin accumulates in the circulation because the liver is unable to excrete all the extra amount produced.

Hepatic jaundice is the result of damage to liver cells which are prevented from functioning effectively or are too few in number if death of the cells has occurred. In this situation the amount of bilirubin produced by the reticulo-endothelial cells is normal but the damaged liver is unable to excrete even this normal amount.

Post-hepatic jaundice is the result of some mechanical obstruction of the bile ducts. Bile is produced and excreted normally by the liver but it cannot enter the intestines because of a block. Pressure in the biliary canaliculi in the liver rises and the bilirubin re-enters and accumulates in the circulation.

## DIAGNOSIS

The first question to be decided must be: Is jaundice present? The child should be examined in good natural light when the yellowness of the skin and conjunctivae will be most apparent. It will be recalled that although the upper limit of the normal level of bilirubin is 0·8 mg per 100 ml clinical jaundice is not apparent until the level reaches about 2 mg per 100 ml.

Jaundice will be quite obvious when it is profound as in congenital obliteration of the bile ducts or in severe haemolytic disease of the newborn, but is by no means so easily detectable in lesser degrees of haemolytic disease, where early treatment may be required, or in acholuric jaundice or in infective hepatitis.

Having decided that jaundice is present, confirmed if necessary by an estimation of the serum bilirubin, the probable cause will then have to be elucidated.

The correct diagnosis depends much more on a careful clinical assessment of the case, taking into account all details, than on relying on a whole battery of pathological investigations.

Distinction between pre-hepatic, hepatic and post-hepatic causes of jaundice should take into account the following. In haemolytic

disease there is always a severe anaemia associated with jaundice. Considerable disturbance of general health is present in most types of pre-hepatic and hepatic jaundice. This is much less in the post-hepatic varieties, at least in the early stages.

The urine is bile-stained where obstruction to the bile passages is present. In pre-hepatic jaundice, unless complicated by liver damage and blockage of the small canaliculi, there are no bile pigments in the urine but an excess of urobilinogen.

The stools will be pale clay coloured in the obstructive type of post-hepatic and hepatic jaundice and will look normal in pre-hepatic jaundice. The presence of bilirubin or stercobilin in the faeces rules out total biliary obstruction.

*Investigations*

In most instances a period of observation will decide the diagnosis but such delay may not be possible. The following tests must therefore be considered, but it must be admitted that they are far from reliable in children and even less so in infants.

The van den Bergh reaction will help to distinguish between predominantly obstructive causes and a predominantly haemolytic jaundice, but it cannot give information on the site or the degree of the obstruction.

The serum alkaline phosphatase is grossly raised in obstructive jaundice but this may also occur in other types of jaundice. The empirical liver function tests are only of limited value. A laparotomy and a liver biopsy may be required to make an accurate diagnosis.

## SIGNIFICANCE

The significance of jaundice depends on the cause. In the newborn one should beware of the diagnosis of 'physiological jaundice'. Consideration must first be given to the possibility of a haemolytic anaemia due to Rh factor incompatibility. The mother's blood group should of course be known but situations may occur when this information is not available. The infant's blood should be grouped and the bilirubin content estimated to assess the need for a possible exchange transfusion. In differentiating the possible causes of jaundice in the newborn it is wise to assume that jaundice in the first 24 hours of life is never 'physiological'.

The longer the jaundice persists the more likely it is to be due to a serious pathological cause. Physiological jaundice begins to fade after the first week and has generally cleared by the fourteenth day of life.

In older children jaundice is rarely a matter of urgency but its accurate diagnosis and management may present many difficulties.

## SPECIFIC CONDITIONS

### GALACTOSAEMIA

Galactosaemia is the result of a deficiency of an enzyme in the red blood cells concerned with the metabolism of galactose. Lack of enzyme results in accumulation of galactose. The clinical features are jaundice due to liver damage, enlargement of the liver, failure to thrive and mental deficiency. The urine contains galactose, albumin and abnormal amino acids. Early diagnosis is essential and treatment by eliminating galactose and lactose from the diet will enable the child to grow up normally. Normal diet can usually be taken once adolescence is reached.

### CONGENITAL OBLITERATION OF BILE DUCTS

There are two pathological types—intrahepatic obliteration of bile ducts and atresia of common bile duct. The latter is curable, but not so intrahepatic atresia. The main clinical feature is jaundice of increasing intensity, which is present from the second day of life, but diagnosis may be delayed till the second or third weeks owing to confusion with physiological jaundice. The urine contains bile and the stools are pale but not completely white. The liver is enlarged and later the spleen as well.

Unless the condition can be corrected surgically death occurs within the first two years. Biliary cirrhosis develops with portal hypertension, splenomegaly and ascites.

### HAEMOLYTIC DISEASE OF THE NEWBORN (ICTERUS GRAVIS NEONATORUM)

Jaundice is one feature of haemolytic disease of the newborn. Other manifestations may be maceration and stillbirth, hydrops foetalis and anaemia.

*Aetiology*

The cause is incompatibility of foetal and maternal blood groups. This incompatibility is most often due to the mother being Rh-negative (D-negative) and the foetus being Rh-positive. The mother becomes sensitized by repeated pregnancies or previous incompatible blood transfusions and produces antibodies against the foetal blood. These maternal antibodies pass across the placenta into the foetal

circulation and damage the red blood cells and other foetal organs.

Jaundice is thus the result of haemolysis of the foetal red cells by the maternal antibodies.

The incidence (2 per 1,000) of clinical haemolytic disease in the newborn is much less than the theoretical expected incidence (10 per 1,000).

*Clinical Features*

Jaundice develops within the first 24 hours of birth. The child is lethargic. Anaemia, which is always present, is masked by the jaundice. In severe cases, where the serum bilirubin is above 20 mg per 100 ml, kernicterus results after 5–9 days with extensive neurological disturbances, such as rigidity, fits and cyanotic attacks.

*Management*

All jaundiced neonates should have the cord haemoglobin and serum bilirubin estimated. If former is low (below 15 g per 100 ml) and the bilirubin high (above 2·8 mg per 100 ml) then exchange transfusion is essential.

## INFECTIVE HEPATITIS

Infective hepatitis is an acute infection caused by a virus. A diffuse hepatitis occurs with focal degeneration of the liver cells. Healing by regeneration occurs in most cases but in some there is a variable extent of fibrosis. Where there has been much damage the eventual outcome may be cirrhosis of the liver.

*Aetiology*

Infective hepatitis is rare under 3 years of age. Epidemic forms occur during wars when young men are crowded together and when spread can also be transmitted by non-sterile syringes or transfusions, etc. (homologous serum jaundice).

There are probably two causal viruses: (1) which causes infective hepatitis with an incubation period of 30 days (this is transmitted by contaminated food or water) and (2) which causes homologous serum jaundice with an incubation period of about 90 days.

*Clinical Features*

Although the illness is infective most cases are seen sporadically, except during major epidemics in closed communities. It is rare for more than one case to occur in a family and rare for the family doctor to be treating more than one case at a time.

The pre-icteric phase lasts 3–7 days, with malaise, nausea, anorexia and vomiting. Fever may be present. There is discomfort in the upper abdomen.

Jaundice appears after a week and may persist for a few weeks. The first signs are yellow eyes, dark urine and pale stools.

*Course*

As a rule the course is benign with complete clearance of jaundice within 2 weeks. Rarely, severe cases occur with a fulminating course of acute liver failure, coma and death. A recurrent and relapsing course is another rare type but with eventual recovery. Following a moderate or severe attack (there may be no correlation between the clinical picture and the pathological damage) the destroyed liver cells are replaced by fibrous tissue and cirrhosis with liver failure may occur.

*Diagnosis*

In the pre-icteric phase the diagnosis is difficult. The condition should be considered in children with a vague digestive illness going on for more than 2 days.

Once jaundice appears differentiation is necessary from Weil's disease, liver poisoning by drugs, haemolytic anaemias and gallstones.

*Investigations*

The urine will contain bile salts and pigments and the stools are pale. The van den Bergh reaction is direct in the early stages and biphasic later but other liver function tests are generally inconclusive.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Neonatal* | | | | |
| Physiological | Within 5 days of birth; clears within a week | Clinical | Nil | |
| Haemolytic disease | Two per 1,000 births; jaundice within 24 hours of birth; anaemia | (1) Mother Rh-negative (2) Serum bilirubin | Exchange transfusion when indicated | Good prognosis if exchange transfusion |
| Galactosaemia | Rare—Jaundice; liver enlarged; failure to thrive; mental retardation | Urine | Diet | Fair outlook with therapy |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Toxoplasmosis | Rare—Jaundice; choroidoretinitis, hydrocephalus or microcephaly; spleen and liver enlarged; lymph glands enlarged; rashes | Toxoplasmin skin test | Nil specific | Doubtful course |
| Obliteration of bile ducts | Rare—Present at birth and deepening jaundice | Clinical and laparotomy | Surgery | Depends whether amenable to surgery |
| *Infancy* | | | | |
| Acholuric jaundice (congenital haemolytic anaemia) | Spherocytic haemolytic anaemia; jaundice; spleen enlarged; gall-stones | (1) Blood picture<br>(2) Urine | Possible splenectomy | Doubtful outlook |
| Infective hepatitis | Rare under 3 years of age<br>Obstructive type of jaundice | Clinical | Nil specific | Good on whole |
| *Childhood* | | | | |
| Acholuric jaundice | Crises occur in childhood | (1) Blood<br>(2) Urine | Nil specific | |
| Sickle-cell anaemia | In Negroes; haemolytic anaemia | Blood | Nil specific | |
| Infective hepatitis | Virus infection; frequent type of jaundice; preceded by nausea and vomiting; obstructive type; incubation period 1 month | Clinical | Nil specific | Good in great majority |
| Homologous serum jaundice | Virus infection, transmitted by injections; clinically similar to infective hepatitis; incubation period about 3 months | Clinical | Nil specific | Good in great majority |
| Weil's disease | Leptospiral infection; jaundice; kidney involvement and haemorrhages | (1) Blood culture<br>(2) Urine | Antibiotics | Fair outlook |
| Poisons and drugs | For instance, chloroform, phosphorus and carbon tetrachloride | Clinical | Nil specific | Fair outlook |
| Bile-duct obstruction | For instance, gall-stones, new growths and glands, obstructive jaundice | (1) Clinical<br>(2) Radiograph<br>(3) Laparotomy | Surgery | Depends on cause |

# LIMP

A limp in a child must always be taken seriously and only after exclusion and consideration of possible serious causes can the parents be reassured. Even then the decision to dismiss the case as insignificant must never be taken as the result of a single examination. A follow-up and reassessment over a period of time are always necessary.

## CAUSES

*Hip*

*Infancy*—In infancy the causes of limping connected with hip conditions include trauma, transient synovitis, congenital dislocation of the hip, tuberculosis, acute suppurative arthritis, rickets.

*Childhood*—In childhood causes include transient synovitis, trauma, osteochondritis (Perthes' disease), tuberculosis, slipped epiphysis.

*Knee*

Knee conditions giving rise to limp include trauma, synovitis (non-specific, rheumatic, rheumatoid), tuberculosis, osteochondritis (Schlatter's disease).

*Ankle*

Ankle conditions include trauma, osteochondritis, tuberculosis.

*Foot*

Foot conditions include ingrowing toenail, chilblains, plantar warts, corns, blisters, claw foot and claw toes, ill-fitting footwear, march fracture, osteochondritis of metatarsals (Freiburg's disease), osteochondritis of navicular (Köhler's disease), osteochondritis of calcaneum (Haglund's disease), calcaneal bursitis.

*Spine*

Spinal conditions producing limp include tuberculosis and scoliosis.

*Central Nervous System*

Central nervous system disorders producing limp include poliomyelitis and spasticity (various types).

*Muscles*

Myopathies of many kinds may produce limping in children.

*Psychological Disturbances*

Psychological disturbances must be borne in mind as possible causes of limping when no other cause is apparent.

## PATHOLOGY

The long list of possible causes shows that a limp can be the result of some abnormality to any part of the weight-bearing structures from the spine through the hips, knees, ankles and feet. Any condition causing pain, weakness or asymmetry of the weight-bearing apparatus can cause a limp.

## DIAGNOSTIC PROCEDURES

*History*

The history is of the greatest importance. Different conditions will be considered at different ages. The duration of the limp is of help. Congenital lesions, for example, such as congenital dislocation of the hip, will be present from the time the child begins to walk and it is usually only after some delay that the mother will seek medical advice. Many other conditions also occur slowly over a time, for instance tuberculosis and osteochondritis. Much more dramatic and sudden are painful conditions related to trauma and acute inflammation.

*Examination*

Examination must follow a pattern. The limp must be observed and an attempt made, at least, to localize the possible cause, possibly in the hips, knees or feet, etc. The child should then be examined naked, standing and lying. The spine and the individual joints of the lower limbs must be examined and then the feet, muscles and central nervous system.

Localization of a possible cause will usually require a radiograph of the particular joint and bones. In addition blood examinations including the erythocyte sedimentation rate will be helpful in infective and inflammatory conditions.

## SIGNIFICANCE

A limp must always be taken seriously and all the conditions mentioned should be considered in the differential diagnosis. Many limps are transient and self-correcting. Of serious importance are persistence for more than a few days and association with definite abnormal physical signs.

The specific conditions that may cause a limp are most conveniently considered according to the age periods at which they may occur.

## SPECIFIC CONDITIONS IN INFANCY

(Age: Birth—2 years)

There is no normal age at which a child learns to walk. Individual variation is considerable and walking may begin at any time between 9 months and 2 years. Delay is not necessarily evidence of physical disease or mental retardation (although either may be present). Once walking has started various habits such as in-toeing may develop, due to the temporary incoordination. These 'normal abnormalities' in walking development must be recognized and distinguished from organic conditions.

*Congenital Dislocation of the Hip*

*Clinical features*—To achieve good results from treatment the orthopaedic surgeon must be allowed to start treatment early. The condition must therefore be diagnosed at the earliest possible moment, even before the child has started to walk. The only indication of abnormality may be slight alteration in shape or shortness of one leg. Often the mother notices a difference in the level of the gluteal folds or creases or inner sides of the thighs. Such observations by a mother should never be dismissed merely as over-anxiety, nor should reliance be placed on clinical examination alone. Radiographs must always be taken.

Limp is often the first abnormality to be noticed, for it is rare for a diagnosis to be made beforehand.

The limp is of the Trendelenburg type and is painless. Since the gluteal muscles cannot abduct the hip, because of lack of stability of the head and lack of leverage, the trunk rolls across the supporting foot at each step. (The Trendelenburg test, consisting of dropping of the pelvis towards the weight-bearing leg is positive.)

*Aetiology*—The cause is probably an error of development in early

foetal life. It has also been suggested that congenital dislocation of the hip is due to abnormal intra-uterine pressure. There is sometimes a family history of the condition.

Epidemiologically, the incidence is greater in some districts than in others, for example in certain parts of France and Italy.

Females are affected much more often than males, the proportion being about 7 to 1. Unilateral dislocation is more common than bilateral dislocation. When both hips are affected the gait has a characteristic waddling character.

*Acute Arthritis of the Hip*

Acute arthritis of the hip is rare and usually results in a severe illness with fever and extreme pain and immobility of the limb, but mild cases may occur with a painful limp as the only sign. The latter type must be distinguished from a tuberculous arthritis.

## SPECIFIC CONDITIONS IN CHILDHOOD

(Age: 2–7 years)

*Transient Synovitis*

Transient pain in the hip or knee joints with a limp must always be regarded seriously until proved otherwise. Although transient symptoms are common and account for probably more than 4 out of every 5 cases in young children, it is always advisable to have a radiograph examination, with a repeat in a short time (2–3 weeks) if symptoms persist.

The causes of these transient troubles are uncertain. It is possible that many are the results of minor trauma or a mild non-specific infection of the joint.

*Tuberculosis*

Although it is becoming increasingly rare, tuberculosis of the hips and knees must still be considered as possible causes of a persistent limp in early childhood.

Most cases occur within the first decade, and more than half between 3 and 5 years of age and are secondary to a focus of infection elsewhere.

The onset is insidious. At first the limp is present only when the child is tired and may disappear after rest. Pain soon follows. When the hip is affected the pain is usually referred to the front of the thigh and the inner side of the knee. The movements are all limited and the hip is held in slight flexion, abduction and external rotation.

Spasm, deformity, swelling and wasting are all late signs. At the knee swelling occurs early and is associated with muscle wasting and a flexion deformity. The tuberculin test will be positive.

*Perthes' Disease of the Hip*

This can occur at any age from 3 to 16 years, but the maximal incidence is from 4–9 years. It is an osteochondritis affecting the head of the femur. During the active stage there is softening with deformity resulting in flattening of the femoral head. Treatment is aimed at preventing this deformity. Spontaneous healing is the rule with laying down of new bone.

The first complaint is of a limp. Pain is not usually marked. Examination shows muscle spasm, and in particular restriction of abduction, extension and external rotation.

A radiographic examination is essential in diagnosis. The earliest changes are an increase in the joint space and increasing density of the epiphysis. Later fragmentation of the epiphysis with flattening of the head occurs.

*Spasticity*

Although severe forms present gross features with spastic limbs, 'scissor limbs' and athetoid movements of the hands, the mild case may be missed until walking is attempted, usually in the third year. In fact the gait may appear as a limp if the condition tends to be unilateral.

*Poliomyelitis*

A missed case of poliomyelitis may first present as a child with a limp and it is only then that wasting of the calf or thigh muscles becomes apparent.

*Osteochondritis of the Navicular*

Osteochondritis of the navicular (Köhler's disease) occurs most often between 4 and 6 years of age. The child complains of pain in the inner side of the foot and walks with a painful limp. Examination shows tenderness and swelling over the navicular. A radiograph confirms the diagnosis. There is no need for any treatment as in the case of Perthes' disease.

## SPECIFIC CONDITIONS IN OLDER CHILDREN

(Age: 7–15 years)

*Slipped Femoral Epiphysis*

This is an abnormal condition of the upper femoral epiphysis

resulting in displacement of the head on the neck of the femur. The condition is more frequent in boys (usually fat children), and is most frequent between 12 and 15 years.

Referred pain to the thigh or knee is the first symptom. Limp due to muscle spasm develops as pain increases. On examination the only findings in the early stages are muscle spasm and limitation of abduction. Later, when gross slipping has occurred, limp is marked and there is an adduction deformity as well as spasm.

*Osteochondritis of Calcaneum and Metatarsals*

Osteochondritis of calcaneum (Haglund's disease) and metatarsals (Freiberg's disease) occurs at this period. Pain is referred to the heel or the foot (second metatarsal heads are affected). The diagnosis can only be confirmed by radiographs.

*Pain in the Feet*

Pain in the feet as a cause of limp may be the result of a number of relatively minor conditions. The gross deformities such as hallux valgus, pes cavus and flat feet are adult conditions.

*Plantar Warts*

Plantar warts are probably the most frequent cause of a painful foot causing a limp. They occur over the heels and forefeet. Their appearance is characteristic, consisting of circular areas with a dark punctate surface.

*Calcaneal Bursitis*

Calcaneal bursitis occurs round the insertion of the tendo achillis. It presents as a red swollen area. It is most common in adolescent girls.

*Ingrowing Toenail*

An ingrowing toenail only causes pain when it becomes secondarily infected.

*March Fracture*

A march fracture of a metatarsal can occur in adolescent boys who go on a long hike during their holidays. Pain is referred to the anterior metatarsal region and there is tenderness, accurately localized, to some part of the shaft of the affected metatarsal. A radiograph is necessary to confirm the diagnosis.

## LIMP

### TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Hip<br>Trauma<br>Transient synovitis<br>Congenital dislocation<br>Tuberculosis<br>Acute arthritis<br>Rickets<br>Perthes' disease<br>Slipped epiphysis | In considering the child with a limp a systematic approach is necessary and one must first decide on the site of the lesion and then consider possible causes | Diagnosis will depend on the condition but is based on clinical assessment and radiological appear-ances | Depends on primary condition | Depends on primary condition |
| Knee<br>Trauma<br>Synovitis<br>Tuberculosis<br>Osteochondritis | | | | |
| Ankle<br>Trauma<br>Osteochondritis<br>Tuberculosis | | | | |
| Foot<br>Ingrowing toenail<br>Chilblains<br>Plantar warts<br>Corns<br>Claw foot<br>March fracture<br>Osteochondritis<br>Calcaneal bursitis | | | | |
| Spine<br>Tuberculosis<br>Sclerosis | | | | |
| Central nervous system<br>Poliomyelitis<br>(late effects)<br>Spasticity | | | | |
| Muscles<br>Myopathies | | | | |

# NASAL OBSTRUCTION, CATARRH AND MOUTH BREATHING

These symptoms are so common in young children that it is often tempting to ask the question whether the symptoms are really abnormal? They are nuisances and closely related to the whole general condition of the child and his environment.

## CAUSES

(1) The 'catarrhal child' syndrome, which includes these symptoms among others. The causes are related to infection and immunity but also to social and environmental factors. (See chapter on Cough.)

(2) Enlarged adenoids, which cause postnasal obstruction and are really part of the 'catarrhal child' syndrome.

(3) Vasomotor rhinitis, which is an allergic state of the nasal mucous membrane in which it swells in response to a variety of exogenous and endogenous stimuli.

(4) Nasal polypi, which only tend to occur in children who suffer from vasomotor rhinitis.

*Rare*

(5) Intranasal foreign bodies, which usually cause unilateral obstruction with discharge.

(6) Nasal diphtheria, which may occur as a subacute infection with bloody unilateral discharge.

(7) Congenital posterior choanal atresia, which is a very rare genetically inherited condition present at birth. If bilateral, the infant may die because mouth breathing may not be possible. If unilateral, the only features may be feeding difficulties, attacks of cyanosis and dyspnoea and unilateral nasal discharge.

(8) Neoplasms, which are excessively rare in children. Secondary invasion from bony tumours may occur.

## PATHOLOGY

Persistent nasal obstruction may lead to:

(1) Abnormal facial appearance. The so-called 'adenoidal facies', which may, however, be a normal phase in development.

(2) Defective development of the teeth.

(3) Infection of the sinuses, ears, and chest, because of failure of postnasal drainage.

## DIAGNOSIS

Assessment must take into account the many factors that influence the 'catarrhal child' syndrome; for instance, age—it is troublesome at 3–7 years, and social class—it is present at all social levels but the more severe grades occur in lower social groups.

*Examination*

Examination of the whole child is necessary. Account must be taken of the general appearance and development of the child in relation to the mother's history. Most often the child looks fit and well in spite of a history of troublesome nasal obstruction.

Since it is part of a syndrome, it is also necessary to examine the ears, fauces and chest as well as the nose. However, apart from the stuffy nose with its congested mucosa and palpable cervical glands little of note is usually found. The size of the adenoids is difficult to assess. Ideally this should be done by examination with a laryngeal mirror, but even then a true assessment is difficult because very large adenoidal pads often cause no symptoms. On the other hand, severe nasal obstruction and catarrh may occur in the presence of very small adenoids. Likewise the size and appearance of the tonsils are of little practical value.

The role of infected sinuses is also difficult to place, for although proof puncture may yield pus, improvement in the condition is by no means certain after sinus infection has been cleared.

Beware of unilateral nasal discharge, especially if it is blood-stained. It generally implies a serious condition such as a missed intranasal foreign body, nasal diphtheria or an intranasal neoplasm.

## SIGNIFICANCE

*Is Nasal Obstruction Present?*

This can best be answered by the mother's story of persistent mouth breathing, especially with snoring at night. Examination will reveal the swollen, red and congested mucosa or perhaps, rarely, some other cause.

*What is its Cause?*

This will have to be decided by means of a clinical assessment,

but by far the most common causes are variants of the 'catarrhal child' syndrome.

Unilateral nasal discharge and obstruction suggest local causes.

*What Should be Done?*

It should be appreciated that the presenting symptoms of nasal obstruction, catarrh and mouth breathing are not caused by simple local causes such as enlarged adenoids. They are part of a wider syndrome in which local treatment is but a part.

## SPECIFIC CONDITIONS

### THE 'CATARRHAL CHILD'

In an average year about two-thirds of all children (aged 10 years or under) are taken to their family doctor for advice for variants of this syndrome. Of the others many suffer from recurrent colds and coughs associated with nasal obstruction. It is the most common condition in children.

*Aetiology*

The aetiology is multiple and although infection is the most important, allergy, constitution, climate, environment and emotions must be considered.

*Epidemiology*

There is a definite age pattern. A moderately high level of incidence is observed in the first three years of life followed by a peak at 4–8 years of age and with a marked decline after the eighth year. Boys are more likely to be affected than girls and children with older brothers and sisters suffer symptoms earlier than eldest and only children. As might be expected, the conditions are worse in winter months between October and April.

*Clinical Features and Course*

The main features are the constant stuffy nose with mucopurulent discharge. The nasal mucosa is congested and red, there is a variable degree of adenoid and tonsil enlargement, the cervical glands are palpable and the mother complains of the child snoring at night and of constantly holding his mouth open. The child looks alert and obviously is not ill.

The course is for this state to persist for a couple of years, being worse in winter but also present in the summer, and then there is a

very definite natural tendency for a spontaneous improvement, probably associated with immunological and developmental changes.

### ALLERGY

It is difficult to assess the importance of the allergic element as a cause of nasal obstruction. It is definitely unusual to find evidence of it in infancy but from about the age of 3 years onwards there are some children in whom certain clinical features point towards an allergic cause.

*Clinical Features*

The features are a positive family history of hay fever and asthma, a story of recurrent bouts of sneezing, usually occurring early in the morning after waking and going on for 5, 10 or 30 sneezes one after the other. Another characteristic feature in these children is the irritation that they get in their noses, causing them to rub them violently with the palms of their hands.

The nasal mucosa has a slightly different appearance from that in the catarrhal child—it is swollen and has a boggy appearance. In some children there is a definite seasonal pattern as in hay fever, from May until August, but in others the sneezing attacks and nasal obstruction are present on and off all the year.

*Management*

Management is difficult. Antihistamine preparations and desensitization procedures do not produce any dramatic results. Fortunately the tendency in many is towards some improvement with age, but in some the condition will persist into adult life.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Catarrhal children | Common between 3 and 8 years; syndrome of recurrent respiratory infections; variable general upset | Clinical | Symptomatic | Natural tendency to improvement |
| Enlarged adenoids | Part of catarrhal child syndrome | Clinical | Surgical removal in carefully selected cases | Natural tendency to diminish in size after age of 7 years |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Vasomotor rhinitis | Allergy; nasal obstruction and bouts of sneezing; may be associated with asthma and eczema | Clinical | (1) Symptomatic<br>(2) Antihistamines | Natural tendency to improve |
| Nasal polypi | In children with allergic nasal mucosae; nasal obstruction and widening of nose | Clinical | Surgical removal | Recur after removal |
| Foreign bodies | Rare—Often no definite history present; unilateral discharge and obstruction | Clinical | Surgical removal | |
| Diphtheria | Rare—Unilateral blood-stained discharge and obstruction; membrane may be present | Bacteriological | Antitoxin | |
| Choanal atresia | Rare—Congenital; potentially dangerous in infants; bouts of dyspnoea and cyanosis | Clinical | Surgical repair | May be fatal if bilateral, complete and untreated |
| Neoplasms | Rare—Secondary invasion from maxillary tumours; discharge, obstruction and swelling | (1) Clinical<br>(2) Radiograph<br>(3) Biopsy | (1) Surgical<br>(2) Radiotherapy | Bad prognosis |

# NECK—ENLARGEMENT OF CERVICAL LYMPH GLANDS

Lymph glands in the neck are frequently palpable in children. A clinical decision has to be made as to when prominent and persistent enlargement requires further investigations.

## CAUSES

### PHYSIOLOGICAL

Palpable cervical lymph glands are present in three out of every four children between 3 and 8 years of age. This feature is part of the natural hypertrophy of lymphoid tissues that occurs at this period.

### INFECTIONS

Bacterial or viral infections are the most frequent cause of persistent cervical gland enlargement. The site of the enlarged glands depends on the part affected. Upper cervical glands are affected in acute throat infections, submandibular glands in infections of mouth and teeth and occipital glands when the scalp is infected.

*Acute*

*Acute throat infections*—These frequently cause a painful and tender enlargement of the upper cervical glands. If the infection is severe and treatment delayed suppuration and abscess formation may result.

*Glandular fever*—In glandular fever enlargement of the glands in the neck with an infected throat are early features. Enlarged glands will also be found in the axillae and groins and the spleen is often palpable. Diagnosis is confirmed by the mononucleosis and positive Paul–Bunnell test. (See also chapter on Sore Throat.)

*German measles*—In german measles (rubella) the posterior cervical glands are palpable.

*Chronic*

*Recurrent infections of upper respiratory tract*—Recurrent infections of the upper respiratory tract such as tonsillitis, otitis media and sinusitis are part of the 'catarrhal child' syndrome. (See chapter on Cough.)

*Pediculosis capitis and other scalp pyodermias*—Pediculosis capitis and other forms of pyodermia of the scalp are responsible for persistent swelling of occipital lymph glands. Abscesses often form in these glands.

*Rare*

*Tuberculosis*—Tuberculosis of the cervical lymph glands is now a very rare condition in Great Britain because of the universal practice of pasteurization of milk and tuberculin tested herds of cows. The condition presents as painless enlargement of the glands, which are matted together. A cold abscess is a frequent complication together with the formation of a sinus. The tuberculin skin test is positive.

### NEW GROWTHS (RARE)

All these are rare causes of cervical lymph gland enlargement but must be considered in the differential diagnosis of persistent and painless enlargements.

*Leukaemia*—Although enlarged glands in the neck may be an early feature, usually they do not become prominent until the disease is well advanced.

*Lymphosarcoma*—Initially discrete, the glands soon become matted together in a hard mass.

*Hodgkin's disease*—Hodgkin's disease (lymphadenoma) is characterized by an insidious and painless enlargement. At first soft, the glands soon become firm and rubbery. Although discrete, their increasing size may cause them to adhere.

*Secondary malignant deposits*—These are very rare but may occur in cases of neuroblastoma, sarcoma of the maxilla and throat.

### OTHER CAUSES (RARE)

Cervical glands may on rare occasions enlarge as part of a general lymphadenopathy in juvenile rheumatoid arthritis, syphilis, disseminated lupus erythematosus, serum sickness, storage diseases where the reticulo-endothelial system is involved and in haemolytic anaemias.

## PATHOLOGY

Apart from the rare malignant conditions mentioned, swelling of the cervical lymph glands is secondary to some other primary condition and the pathological changes will be those of acute or chronic inflammation.

## DIAGNOSIS

Persistence of enlarged cervical glands in children demands careful investigation.

### EXAMINATION

The area drained by the glands, namely the scalp, face, ears, nose, throat and teeth and mouth, should be carefully examined for sites of primary infection or other lesions.

The physical features of the glands themselves will help considerably in suggesting the cause of enlargement. The physiologically palpable glands are small and discrete and are present in both anterior and posterior triangles of the neck. Acutely inflamed glands are tender, often with oedema and redness of the overlying skin.

Tuberculous glands are initially discrete, firm and painless, but with time become matted together and then soften to form a fluctuant cold abscess with adherence to the skin. Sinuses develop with the breakdown of the skin and secondary infection causes redness, pain and tenderness.

The glands of Hodgkin's disease are classically painless, firm, rubbery and discrete although their large size may cause them to appear fixed and adherent.

In lymphosarcoma the glands are hard, fixed and matted together. In leukaemia the glandular enlargement never reaches a very marked size, the glands tend to be discrete and soft.

### ASSOCIATED FEATURES

The associated features are of importance. There may be fever and the child is obviously ill in glandular fever, Hodgkin's disease, leukaemia and of course in acute infections. The spleen and liver may be enlarged in leukaemia.

### ANCILLARY INVESTIGATIONS

Confirmation of the diagnosis may require a biopsy, a piece of the enlarged glands being removed for histological examination and perhaps for culture, for possible bacteria and fungi, and guinea-pigs may be inoculated in suspected tuberculosis. A tuberculin skin test should be carried out whenever the gland enlargement persists for more than 2 months. White blood cell examinations, bone marrow biopsy, Wassermann and Kahn tests, and a chest radiograph may all be necessary before the diagnosis is established.

## SIGNIFICANCE

The following facts are to be borne in mind:

(1) Palpable cervical glands are present in most young children between 3 and 8 years of age.

(2) Features that suggest the need for further investigations are increasing size, strict localization, severe illness in the child and the presence of other suspicious features either in the glands themselves or elsewhere, for example, fever, palpable spleen, anaemia and a haemorrhagic tendency.

(3) Glandular enlargement is almost always secondary to some other cause which has to be defined by suitable procedures.

## SPECIFIC CONDITIONS

### LEUKAEMIA

Relatively more frequent now than in the past, leukaemia is still a very rare disease. The average general practitioner will see only about one new case every 7–10 years. The average paediatrician will have about 3–6 cases under his care at any one time. In children it occurs more often in infancy and is then infrequent until adolescence. Acute lymphatic leukaemia is the most usual variety.

*Clinical Features*

Pallor and lassitude are the most frequent presenting features. Spontaneous bruising and petechiae occur. Lymph glands enlarge, and the spleen becomes palpable. Secondary infection of the throat with stomatitis and fever are late features. Blood examination shows a normocytic and normochromic anaemia. The white cell count is generally raised but this may not be high in the aleukaemia phase. Primitive cells are found in the peripheral blood and in the bone marrow.

### HODGKIN'S DISEASE

Hodgkin's disease is uncommon in early childhood. There is an insidious onset with enlargement of glands in the neck as an early sign. The glands are discrete and rubbery and gradually increase in size. The children become ill with fever and eventually die from exhaustion or intercurrent infection.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Physiological | Palpable cervical glands present in more than 75 per cent of normal children during the phase of natural lymphatic hypertrophy | Clinical | Reassurance and observation | Natural regression |
| Acute infections | Painful and tender enlargement following primary throat or skin infections | Clinical | Antibiotics | Good response to therapy |
| Glandular fever | Tender discrete enlargement; sore throat; fever, spleen palpable | Mononucleosis; positive Paul-Bunnell test | Symptomatic | Spontaneous recovery within 2–4 weeks |
| Chronic infections | Tender enlargement of anterior cervical, posterior cervical or occipital glands; primary focus in skin or mouth | Clinical | Treatment of primary cause | Good response to therapy |
| Tuberculosis | Rare—Insidious onset; persistent enlargement; matted glands with eventual fluctuation, discharge and sinus formation | (1) Clinical<br>(2) Positive tuberculin test | (1) Antibiotics<br>(2) Surgery | Recovery possibly with scarred neck |
| Leukaemia | Rare—General illness with anaemia, bruising and bleedings; enlarged soft and discrete glands; palpable liver | (1) White blood cell count<br>(2) Bone and marrow biopsy | (1) Steroids<br>(2) Antibiotics<br>(3) Antimitotics | Fatal |
| Hodgkin's disease | Rare—Insidious enlargement of glands; discrete and rubbery; general illness with fever | (1) Biopsy of glands<br>(2) Chest radiograph for mediastinal glands | (1) Radiotherapy<br>(2) Antibiotics<br>(3) Surgery (rarely) | Fatal |
| Lymphosarcoma | Rare—Insidious and progressive; hard and fixed glands | Biopsy of glands | (1) Radiotherapy<br>(2) Surgery (rarely) | Fatal |
| Secondary malignant glands | Rare—Insidious; hard and fixed glands | Biopsy of glands | Symptomatic | Fatal |

# NERVOUS CHILDREN

The nervous child presents in general practice under many guises and disguises and it is of importance that correct management be based on an accurate causal diagnosis and a proper appreciation of the problems and background in each child and family.

## CAUSES

The 'causes' concerned with the development of the various conditions recognized in nervous children are related individually to the child, his parents and the environment.

### THE CHILD

Each child, even from the neonatal period, is an individual with his, or her, own personality and constitutional make-up. Realization of the differences of these personality traits is fundamental to the understanding of children. As the child develops, these differences become apparent in feeding, sleeping, social contacts and in responsiveness to various routines of management.

Physical disabilities are likely to produce psychological reactions and this is particularly so when they affect the sense organs of hearing and sight. Severe handicaps and repeated illnesses interfere with education and social contacts and cause potential difficulties in the relationships between the affected child and his parents and siblings.

Mental disabilities causing retardation, particularly if this is of so slight a degree that the child is considered normal and attempts are made to treat him and educate him as such, are very likely to cause reactions.

### THE PARENTS

Parental attitudes are a most potent cause of behaviour and allied problems in children. Over-protection and anxiety, lack of love and security, favouritism and rejection, lack of routine and failure to encourage independence in the child are examples of situations that may lead to conflict. The relationships between mother and

child are so close that sometimes she presents the child as a guise for her own emotional problems.

### THE ENVIRONMENT

A stable and united family is ideal for the developing child but even when this exists certain environmental factors predispose to nervous troubles in the child. The position of the child in the family, separation in early life and problems at school are all factors likely to lead to emotional upsets.

## PATHOLOGY

The effects of such stresses on a vulnerable child will vary depending on the child and the situation. Thus management difficulties, behaviour problems or a variety of pseudo-specific symptoms and somatic reactions may result.

It is well, however, to keep the situation in perspective and to remember that all children have behaviour problems at some time or other; that problem children are children with problems, and that these problems are rarely isolated but a part of a general disturbance; finally, parents almost never wilfully upset their children —mismanagement is usually due to ignorance.

## DIAGNOSIS

Faced with the problem of the nervous child the aims should be not merely to exclude organic possibilities and attach the label of 'emotional upset' but rather to determine the various causes in the child, the parents and the environment with a view to helping the family.

Whatever the problem, it is unlikely to be solved by any psychological sleights of hand by the 'experts'. The problems are so frequent and widespread that family doctors and all who are responsible for the care of children must play their part. The approach to the situation must first be one of accuracy in diagnosis followed by correct management. Nothing can replace the clinical importance of a good history supported by one's own personal knowledge of the family and supplemented by a clinical examination.

The busy surgery and clinic are no places for history taking. Adequate time is essential if anything of value is to be achieved. The mother should be seen first alone by appointment with at least half an hour to spare. The mother should be allowed to tell her story of the problems and of the development of the child from birth, and

of her husband's attitude from the time of conception. A note must be made of the times when the milestones were passed. The history must aim at achieving more than a record of information. It should also serve to establish contact and a relationship with the mother.

It is equally important to establish contact with the child and this may be done during the process of examination which must, however, extend beyond the usual clinical procedures to include watching the child at play; in this way information may be obtained on the child's attitude to his problems.

## SPECIFIC CONDITIONS

A large number of almost specific nervous syndromes exist. The most frequent and important are feeding problems (see page 101), sleeping difficulties, excessive crying, enuresis, compulsive habits and truancy and school phobia in older children.

### SLEEP PROBLEMS

The majority of children develop sleep problems in their first 4 years. The patterns comprise: crying when put to bed; refusal to lie down; refusal to allow parent out of the room; awakening and playing in the middle of the night; prolonged failure to go to sleep; sleep rituals; early morning wakening; getting into bed with mother.

The most usual cause is with the parents. Parents often have a wrong idea of the amount of sleep that their baby requires. It is usually an overestimate. Over-rigidity of routine or alternatively failure to achieve a routine are other causes, as are over-anxiety and domination, sharing the bedroom with the parents or such gross errors of management described under 'causes'.

### EXCESSIVE CRYING

Although crying in babies is most commonly due to minor causes such as hunger, physical discomfort, loneliness and flatulence, care must be taken to differentiate the organic illnesses.

As a rule crying from an illness occurs as a deviation from normal patterns. The baby crying from minor nervous causes tends to do so repeatedly many times during a day. The organic illness comes out of the blue and leads to a break in the normal routine pattern. The mother is able to sense that something unusual has occurred. Other features are present such as fever, obvious pain, cough and running nose, vomiting and so on.

A clinical examination is always necessary and particular attention should be paid to the ears and the urine, since otitis media and pyelitis often occur silently in babies.

## ENURESIS

Enuresis is a common condition. It can occur during the night or during the day.

Most cases are due to physiological slow development of bladder control or to psychological causes. Only a very small minority are caused by organic lesions, such as congenital abnormalities of the urinary tract, urinary infections, diabetes mellitus or diabetes insipidus, chronic renal failure, neurological lesions or mental deficiency.

It is important to pick out these organic types. These children are usually incontinent day and night, they look ill, other congenital abnormalities may be present, the stream is poor, the urine is infected or otherwise abnormal and the intravenous pyelogram is abnormal.

The nervous or physiological type may date from birth, the child never having been dry at night. On the other hand there is the 'onset' group where the enuresis does not start until 3–5 years of age following some incident such as a new sibling, admission to hospital, an illness or some other upset in routine. Emotional stresses are not usually obvious and a family history is often present.

Although there is a tendency for the condition to clear up with age some children have persistent enuresis till adolescence. Much can be done to help them and their families by understanding their personal problems and by giving them the opportunities to discuss them.

## SCHOOL PHOBIA AND TRUANCY

### *School Phobia*

School phobia denotes the child's refusal to go to school related to a fear of separation from the parents. It can occur at any age and in all social groups. As may be expected it tends to affect chiefly only-children, or those born many years after their siblings. The child is generally timid, sensitive and spoilt, with other symptoms of nervousness. The parents tend to be indulgent, over-anxious and over-protective and inconsistent and vacillating. The problem is a complex one, depending on the interaction of the child's personality, the parents' attitudes and on the environment at school, where bullying and fear of teachers may be important.

*Somatic symptoms*—Somatic symptoms such as vomiting, headache and abdominal pains are another group of manifestations that appear in young children soon after starting school.

*School Truancy*

School truancy occurs most often at a later age group, from 10–14 years, and represents a symptom of the child's insecurity. It must be regarded as a call for help.

# SORE THROAT

Sore throat is a readily definable symptom when the child is mature enough to be able to complain. In infants and young children, however, acute throat infections tend to present as general disturbances with high fever and malaise, and pain, if present, is often referred to the abdomen, limbs and head.

## CAUSES

An aetiological classification is possible because there is a correlation between the clinical appearances and the causal organisms.

(1) Haemolytic streptococci account for approximately a third to a half of all acute throat infections in children.

(2) Non-bacterial throat infections. No specific cause can be identified in a large group of cases that are probably caused by viruses. Although with our present techniques it is only rarely that a viral cause can be proved it is very likely that with improved diagnostic methods a viral aetiology will be confirmed in this group which accounts for over a half of all acute throat infections. Recently viruses of the Coxsackie group have been isolated from such cases.

(3) Secondary infection of the throat may occur in glandular fever, leukaemia and agranulocytosis.

(4) Vincent's angina, caused by infection with spirochaetes and fusiform bacilli, is associated with oral and dental sepsis. It is not common.

(5) Infection with the fungus *Candida albicans* may either be part of a more generalized infection of the mouth or it may be localized to the tonsils. It is not common.

(6) Diphtheria is now a very rare cause of sore throat but must always be considered in the differential diagnosis.

(7) Poliomyelitis may start clinically with a sore throat and fever. (See chapter on Headache.)

## PATHOLOGY

The infection although primarily affecting the tonsils is never completely confined to them. The adjacent pharyngeal tissues and soft

palate are usually involved also. Spread more deeply to the peritonsillar spaces producing a peritonsillar abscess, or a 'quinsy', is very rare in children.

Severe and persistent infections of the throat must always raise the rare possibilities of diphtheria or secondary infections in leukaemia, agranulocytosis or glandular fever.

## DIAGNOSIS

The diagnosis of the type of sore throat should be clinically and be supported where necessary by simple investigations. Bacteriological examination of a throat swab is essential in all cases that are not settling within 48 hours of observation.

*Examination*

*Local appearances*—The fauces will show three main abnormalities —swelling, redness and exudate. In streptococcal infections the inflammation tends to be localized to the tonsils, which are red and swollen with a discrete follicular type of greyish exudate. In some instances the inflammation extends to involve the soft palate and the pillars of the fauces. A similar appearance may be found in children from whose throat swabs no specific organisms can be isolated; these are presumed to be caused by viruses. In herpangina multiple discrete and small ulcers are present on the fauces and tongue.

In Vincent's angina there are one or two ulcers in the tonsils. These ulcers are necrotic with a very offensive smell.

In candidiasis the posterior pharynx is covered with discrete and superficial white patches without very much underlying redness and swelling. Even now, when immunization has reduced the number of cases to few each year, diphtheria should always be considered in an ill child with a sore throat. Characteristically there is a grey slough which is adherent to the underlying tissues.

Where sore throat is secondary to a condition such as glandular fever, leukaemia or agranulocytosis the fauces are red and covered by a dirty gelatinous exudate with an offensive smell.

*General condition and investigations*—The general condition of the child depends on many factors, such as the virulence of the infection and the general resistance. As a rule the child is most acutely ill with a streptococcal infection and seems little affected in a Vincent's angina. The child is most severely ill when the throat condition is secondary to leukaemia or agranulocytosis.

There is no need for routine throat swabs, although this is the ideal. This must be done, however, in all cases that are not settling

within 48 hours. In approximately a half of all cases of sore throat in children haemolytic streptococci will be isolated. In most of the remainder no pathogenic organisms can be found. Only rarely are candida and Vincent's organisms identified. An examination of the white blood cells is also indicated in cases failing to respond. Only thus can the diagnosis of glandular fever, leukaemia or agranulocytosis be excluded.

## SIGNIFICANCE

Faced with a child with a possible sore throat the doctor must ask himself: Is there a throat infection present? What type is it? What should he do?

*Is there a Sore Throat?*

Acute infections of the throat are rare in infancy and when they occur the condition presents as an acute general disturbance with a high fever and abdominal pain.

Acute infections of the throat increase in frequency from the age of 3 years onwards and reach a peak in the first years at school. It is only by routine examination of the throat of all sick children that such cases will be diagnosed.

*What Type of Infection?*

Haemolytic streptococci and relatively benign viruses account for around 95 per cent of all throat infections in children. However, the other 5 per cent contain the most serious types, those associated with leukaemia and agranulocytosis.

It is not possible to make an aetiological diagnosis on clinical grounds and in case of doubt further investigations must be carried out.

Any child with a throat infection that is not improving within 48 hours should be further investigated by bacteriological examination of a throat swab and an examination of the white blood cells.

*What Should be Done?*

Not all cases of throat infection require antibiotics—some 75 per cent will settle without these specific measures.

## SPECIFIC CONDITIONS

### STREPTOCOCCAL SORE THROAT

The haemolytic streptococcus is one of the most common of pathogens and the throat its favourite site of infection.

*Epidemiology*

*Age*—Children over 5 years are more likely to suffer from acute streptococcal sore throats than those below this age. An acute streptococcal throat infection is rare in infancy. The peak period is during the first few years at school.

*Season*—Contrary to the situation in most other respiratory tract infections, acute throat infections are no more common in the winter months. Epidemics of haemolytic streptococcal infections often occur during a hot dry and dusty summer.

*Clinical Features and Course*

The case often presents as an acutely ill and feverish child who is flushed and vomiting and complaining of abdominal pain and only incidentally or not at all of a sore throat. Examination of the throat is an essential routine in all children and in this case will reveal red and swollen tonsils with a follicular exudate. A scarlatiniform rash, with a diffuse erythema on the trunks, upper limbs and face, occurs in a minority of children. Desquamation of the skin follows one or two weeks later.

The usual course is for improvement to start within 2 or 3 days from the onset and for clinical recovery within 5 days. Recurrences are not uncommon.

*Complications*

Complications are unusual. These may be local such as cervical adenitis, rarely with abscess formation, otitis media and sinusitis.

Remote complications, though rare, are of great importance. Rheumatic fever is said to follow less than 1 per 1,000 of first attacks of streptococcal throats. Acute nephritis occurs in about 1 per 500 of streptococcal infections and is much more likely where the causal streptococci belong to types 4 and 12.

## NON-BACTERIAL THROAT INFECTIONS

The clinical appearances are very similar to those in streptococcal cases and differentiation can be made only on the results of bacteriological examinations of throat swabs.

Herpangina is an acute epidemic febrile illness caused by one of the Coxsackie enteroviruses and occurs in the summer, and here the sore throat is due to the presence of many small and discrete ulcers on the soft palate, pillars of the fauces and tongue.

## GLANDULAR FEVER (INFECTIOUS MONONUCLEOSIS)

*Epidemiology*

Glandular fever is uncommon under 5 years of age. It generally affects adolescents and young adults (15–25 years). On average, a general practitioner may see one or two new cases a year. It is probably a virus infection of very low infectivity and it is extremely rare to see more than one case in a family. Even in boarding schools 'epidemics' are rare.

*Clinical Features*

Three presenting features are a severe sore throat, generalized enlargement of lymph glands and fever.

The tonsils are always affected, with redness, swelling and a thin grey exudate. Lymph glands are enlarged and tender and those in the neck, axillae and groins are most frequently affected. The spleen is often palpable. Fever is generally a marked feature and tends to persist for some weeks. The patient often presents as a 'pyrexia of unknown origin'. Other features such as jaundice, rash, meningism and encephalitis are rare.

*Diagnosis*

Diagnosis can be readily confirmed by examination of the white blood cells. A characteristic mononucleosis occurs. The heterophile antibody reaction (Paul–Bunnell test) when positive is pathognomonic of glandular fever, but cases do occur with identical clinical features and mononucleosis where the test is negative. This negative result should not rule out the diagnosis.

*Course*

Cases vary in severity. Some are almost subclinical in their mildness and are not ill enough to be confined to bed. Others are extremely ill. There is no specific treatment. No deaths have ever been recorded in children. On an average the acute phase lasts 2–3 weeks but a general debility may persist for some months.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Haemolytic streptococcal sore throat | Acute onset; severe general upset; follicular tonsillitis; cervical glands tender; erythematous rash | Throat swab positive | (1) Symptomatic (2) Antibiotics in selected cases (penicillin) | Recovery 3–5 days; acute nephritis and rheumatic fever are possible complications |
| Non-bacterial sore throat (virus) | Acute or subacute onset; variable general upset; greyish exudate on tonsils; cervical glands enlarged | Throat swab negative; diagnosis by exclusion | Symptomatic | Recovery in 3–5 days |
| Glandular fever | Insidious onset; dirty grey exudates on tonsils; lymph glands and spleen palpable | (1) Mononucleosis (2) Paul-Bunnell test positive | Symptomatic | Slow recovery over 2–4 weeks |
| Leukaemia and agranulocytosis | Insidious onset; very ill child; 'dirty throat'; other clinical features, such as enlarged spleen and lymph glands | White blood cell examination | Specific therapy in hospital | Bad prognosis |
| Vincent's angina | Subacute onset; little general upset; deep ulcers on tonsils; oral foetor | Throat swab | Antibiotics (penicillin) | Recovery in 5–7 days |
| Candida infection | Subacute onset; variable general upset; white patches on tonsils and fauces | Throat swab | Nystatin | Recovery in 3–5 days |
| Diphtheria | Acute or subacute onset; severe upset in non-immunized children; adherent membrane; cervical glands enlarged | Throat swab | Antitoxin | Serious disease with high mortality rate |
| Poliomyelitis | Subacute onset; headache and stiff neck; paresis within a few days in paralytic cases | Clinical | Admission to hospital | Serious disease with high mortality rate |

# VOMITING

The causes of vomiting are so poorly understood in many conditions that classification related to physiological mechanisms is not possible. One based on the most important clinical conditions at different age periods is most useful in practice.

## CAUSES

*Newborn*

*Physiological vomiting*—Physiological vomiting at, or soon after, birth is a normal process by which swallowed blood and amniotic fluid are removed. It may occur once or twice but is never the explanation of repeated vomiting.

*Congenital oesophageal obstruction with, or without, fistulae*—Symptoms begin shortly after birth. Excess mucus in the baby's mouth and throat should suggest this possibility. Prompt regurgitation, choking and perhaps cyanosis occur with first feeds. Urgent diagnosis is necessary for successful surgical treatment.

*Intestinal obstructions*—The ileum is the most common site of atresia. Symptoms of obstruction begin in the first 24 hours after birth. The baby may pass greyish mucoid meconium. If the obstruction is proximal to the ampulla of Vater the stools, but not the vomitus, contain bile. Distension is confined to the epigastrium and gastric peristaltic waves may be seen. Radiographs show some air in the stomach and duodenum but more in the small intestine. If the obstruction is distal to the ampulla of Vater the stools do not contain bile but the vomitus does. Abdominal distension is generalized if the obstruction is in or distal to the jejunum. An x-ray of the abdomen in an upright position is essential for diagnosis.

*Imperforate anus*—Symptoms of obstruction also begin in the first 24–36 hours of life with abdominal distension and vomiting. No meconium is passed. The obstruction may be high or low but inspection of the anus may be misleading as it may appear normal when the obstruction is high in the rectum. (See chapter on Constipation.)

*Meconium ileus*—In meconium ileus symptoms of obstruction occur in the first 48 hours and may represent the earliest stage of fibrocystic disease of the pancreas. Meconium if passed is very thick tenacious and tarry.

*Cerebral birth injuries*—Cerebral birth injuries may cause repeated

bilious vomiting. There is usually a history of difficult or precipitate delivery.

*First Week of Life*

*Intestinal stenosis*—As distinct from complete atresia, intestinal stenosis leads to a partial obstruction which may be in the duodenum, small intestine or rectum.

*Hirschsprung's disease*—This may first cause symptoms in the first week of life with slow passage of meconium, vomiting and abdominal distension. (See chapter on Constipation.)

*Malrotation of the bowel and midgut volvulus*—These may also cause obstruction with vomiting in the first week of life.

*Two Weeks to Two Months*

*Pyloric stenosis*—Regurgitation may occur in the first two weeks but true projectile vomiting does not begin until the child is about one month old.

*Feeding difficulties*—Feeding difficulties may lead to vomiting at this stage. Simple errors such as failure to 'bring up wind', too small a teat or underfeeding may be responsible.

*Hiatus hernia with a short oesophagus*—This may cause dysphagia, vomiting and haematemesis. These children are 'very slow eaters' and present many feeding problems.

*Infections*—Such infections as otitis media, tonsillitis, pyelitis (associated with congenital renal abnormalities) and bronchitis and pneumonia are also possible causes of vomiting at this period.

*Cerebral causes*—Cerebral causes are hydrocephalus, subdural haematoma and meningitis. Convulsions usually also occur.

*Six to Eighteen Months*

*Intussusception*—Intussusception occurs from about 12 months onwards and is accompanied by colic and passage of blood-stained mucoid stools. (See chapter on Abdominal Pain.)

*Rumination*—This is rare but a most difficult condition to control.

*All Through Childhood*

*Infections*—In older children the most frequent causes of vomiting are the minor acute infections. These may be local infections of the stomach and intestines with viral and bacterial organisms. Certain foods, drugs such as aspirin or poisons such as lead will also cause vomiting. Distant infections such as otitis media, influenza, pneumonia and pyelitis are also frequently associated with severe and

repeated vomiting. In infective hepatitis nausea and vomiting precede jaundice by a few days.

*Acute abdominal emergencies*—Such acute abdominal emergencies as acute appendicitis, intestinal obstruction and intussusception have to be considered in all children who present with vomiting.

*Periodic syndrome*—The periodic syndrome is now believed to be a psychosomatic disturbance characterized by recurrent bouts of vomiting and abdominal pain. (See chapter on Abdominal Pain.)

*Metabolic disorders*—In disorders such as diabetic ketosis and uraemia vomiting may be an early feature.

*Neurological causes*—Vomiting is one of the clinical manifestations of intracranial neoplasms, even in the absence of any other signs of raised intracranial pressure.

More common causes in the nervous system are migraine and motion sickness, and more rarely subdural haematoma, meningoencephalitis, intracranial abscess and hydrocephalus may be the cause of repeated vomiting.

## PATHOLOGY

Vomiting consists of forceful expulsion of gastric contents through the mouth with vigorous contractions of the abdominal muscles.

The highly co-ordinated sequence of events is controlled by a medullary vomiting centre which lies close to vagal nuclei and the respiratory centre.

Emetic impulses may come in from the labyrinth, pharynx, gastrointestinal and genito-urinary tracts and as a result of various emotional situations.

Repeated vomiting leads to serious fluid and electrolyte disturbances. The main electrolyte lost is the chloride anion. This leads to an increase in carbon dioxide combining power and alkalosis. When diarrhoea and vomiting occur together the loss of cations in the stools may temporarily balance the chloride loss in the vomitus and there may be no change in pH. Starvation and dehydration secondary to vomiting may lead to accumulation of ketone bodies and result in severe ketosis and acidosis. Other disturbances that may occur with vomiting are loss of potassium and general dehydration.

## DIAGNOSIS

Some help in making the diagnosis will come from the type of vomiting. Vomiting that occurs with hiatus hernia or other lesions

of the oesophagus, such as atresia, stenosis or achalasia, is characterized by the infant's inability to take much food at one time and by the prompt return of the food that is swallowed. This food comes up relatively unchanged and undigested with a good deal of mucus. Vomited milk is not curdled.

In gastric vomiting the food is brought up immediately after the meal or perhaps some hours later. Milk is curdled because of the action of the gastric juice, but there is no bile in the vomit. Gastric vomiting may be forceful and projectile (as in pyloric stenosis) or a mere dribble at the side of the mouth in 'posseting'.

Absence of bile in the vomit suggests a complete obstruction proximal to the ampulla of Vater.

Feculent-vomiting occurs in intestinal obstructions. It is a late sign and evidence of a missed diagnosis, either by parents or doctor.

Cerebral vomiting is characterized by its suddenness. There is no preceding nausea. The vomit is sudden, unexpected and projectile.

## SIGNIFICANCE

The significance of vomiting as a symptom varies with the age of the child. Vomiting in the newborn must always be considered as a very serious and urgent matter. The possibility of a surgically remediable obstructive cause in the alimentary tract must always be considered first and steps taken to exclude this by appropriate investigations. Suggestive features are persistent green vomits.

During infancy, until about 7 years of age, the causes of vomiting are more likely to be general than local. Acute gastro-intestinal infections and irritations are frequent and the common childhood infections such as tonsillitis, otitis media and bronchitis are also very often associated with vomiting.

In older children (over 7 years) local lesions such as appendicitis, migraine and acute gastro-intestinal disturbances predominate.

At all these different periods the approach must always be the same. The symptoms must be the sign for a search for a primary cause and appropriate measures taken for its treatment.

## SPECIFIC CONDITIONS

### TRACHEO-OESOPHAGEAL FISTULA AND ATRESIA

So that the child may survive, and there is every hope of survival with reconstructive surgery, the diagnosis must be made early. The malformation is more common than is generally realized and occurs in approximately 1 in every 1,000 births.

Four main types are recognized:

(1) In 75 per cent of all cases the upper end of the oesophagus ends as a blind sac at the level of the thoracic inlet. The lower end of the oesophagus is quite separate from the upper end and is connected with the bronchial tree at the bifurcation of the trachea. Vomiting occurs and at the same time air enters the stomach which becomes distended. Respiratory distress is a prominent feature.

(2) Both ends of the oesophagus end blindly. Vomiting occurs and there is no distension of stomach.

(3) The upper end of the oesophagus is in communication with trachea. The lower end has no connections with trachea or bronchi. Cyanotic attacks with choking on feeding occur.

(4) Both ends of oesophagus communicate with trachea or bronchi. There is vomiting with choking and cyanotic attacks.

The presenting and most characteristic feature is the production of increasing amounts of frothy mucus from the mouth. The abdomen is distended (in the most common type) and this increases when the baby cries. If food is given cyanosis (and death) may occur.

Diagnosis in hospital, by passing a soft rubber catheter which will be held up at 10 cm, is an urgent matter.

### OESOPHAGEAL HIATUS HERNIA

Oesophageal hiatus hernia is a relatively uncommon condition and is associated with a short oesophagus. This shortening is probably secondary to an oesophagitis which is the result of reflux of acid from the stomach.

Symptoms date from birth but become more obvious in the second month of life. Vomiting is the main feature and occurs during a feed. Improvement occurs if the child is fed in an upright position and with more solid foods. Other features are failure to gain weight, and anaemia due to bleeding from the oesophagus.

The most important complication is the development of an oesophageal stricture from the oesophagitis.

### FEEDING TROUBLES

A more frequent cause of vomiting in infancy is underfeeding. Because of the fear of overfeeding or because instructions on the tin of powdered milk are followed too strictly, or because of feeding by the clock, or because of a too small hole in the teat of the bottle the baby will receive insufficient food and become hungry. He will cry, he will swallow air in his distress, and his stomach will become

distended and irritable with reverse peristalsis, causing vomiting. Repeated vomiting in an irritable baby occurring in the first and second months and where no other abnormalities are obvious should make one think of underfeeding.

It is very doubtful whether overfeeding is ever a cause of vomiting.

### PYLORIC STENOSIS

Pyloric stenosis is a not uncommon condition. It is much more frequent in boys than girls (4 : 1) and in the firstborn. It can occur in families and is probably genetically determined. The stenosis of the pylorus is caused by hypertrophy of the pyloric muscles, the circular more than the longitudinal. A 'tumour' results and causes obstruction at the pylorus.

Symptoms first appear about the second to fourth weeks, but diagnosis is often not made until the second month of life.

The vomiting is persistent and characteristically projectile. The vomit is not bile-stained and follows feeds. Loss of weight, dehydration and constipation occur. Visible gastric peristalsis is present and a 'mass' the size of an almond should be palpable behind the right rectus abdominis muscle, midway between the umbilicus and the costal margin.

### MOTION SICKNESS

Approximately 10 per cent of children suffer from motion sickness. The condition is usually first noted in the third year and improves after the tenth year.

### TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| *Newborn* | | | | |
| Physiological | Vomiting of swallowed blood and amniotic fluid; *not* repeated | Clinical | Nil | |
| Oesophageal obstruction | Excess mucus; immediate regurgitation after feed with possible cyanosis | Passage of soft rubber catheter | Urgent surgery | Good with surgical repair |
| Intestinal obstruction | Repeated green vomits; abdominal distension | Radiograph of abdomen in upright position | Urgent surgery | Good with surgical repair |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Imperforate anus | No meconium; repeated vomiting; distension of abdomen; abnormal rectum and anus | (1) Radiograph of abdomen and pelvis (2) Rectal examination | Urgent surgery | Good with surgical repair |
| Meconium ileus | Fibrocystic disease of the pancreas; thick, tarry and tenacious meconium | Radiograph of abdomen | As for fibrocystic disease | Poor prognosis |
| Cerebral birth injuries | Repeated bilious vomits; history of difficult delivery | Clinical | General | Doubtful |
| *First week* | | | | |
| Intestinal stenosis | Partial obstruction with vomiting and distension | Radiograph | Surgery | Good with surgery |
| Hirschsprung's disease | Little meconium passed; distension and vomiting | Clinical and radiological | Eventual surgery | Fair |
| Malrotation and mid-gut volvulus | Distension and vomiting | Clinical | Urgent surgery | Good with surgery |
| *Two weeks to two months* | | | | |
| Pyloric stenosis | Projectile vomiting; pyloric tumour and gastric peristalsis | Pyloric tumour and peristalsis | Surgery | Good with surgery |
| Feeding difficulties | Underfeeding and small teats | Clinical | Correction of errors | |
| Hiatus hernia | Feeding difficulties and vomit | Radiograph | Medical | |
| Infections | Symptomatic of primary disease | — | — | — |
| Cerebral | Symptom of primary disease | — | — | — |
| 6–18 *Months* | | | | |
| Intussusception | Vomit and colic; bloody mucoid stools | Clinical | Surgery | Good with surgery |
| *All ages* | | | | |
| Infections | Gastric and intestinal infections–Hepatitis, otitis media, pyelitis, pneumonia and influenza | Clinical | Specific or symptomatic | Good |
| Abdominal emergencies | Appendicitis, intussusception and intestinal obstruction | Clinical | Surgery | Good |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Periodic syndrome | Psychosomatic; abdominal pain; vomiting | Clinical | Symptomatic and psychotherapy | Good |
| Metabolic | Uraemia and diabetes mellitus | Biochemistry | That of the primary condition | — |
| Central nervous system | Motion sickness; migraine; neoplasms; meningitis; subdural haematoma and hydrocephalus | Various | That of the primary condition | — |

# WHEEZING

The child with attacks of wheezing in the chest is a frequent problem. Approximately 20 per cent of children suffer one or more bouts of wheezing during their first 10 years. It is very important to understand the nature, course and outcome of the causative conditions because the correct management of the child and of the parents depends on this understanding.

## CAUSES

(1) Acute wheezy bronchitis. Many children pass through a phase when they react to infections of the lower respiratory tract with bronchospasm and wheezing.

(2) Asthma. Differentiation from acute wheezy bronchitis is difficult. Diagnosis is most likely to be correct when there is a positive family history.

*Rare*

(3) Inhaled foreign bodies as a cause must be considered when wheezing begins suddenly and is unilateral.

(4) Pressure from enlarged mediastinal glands.

(5) Fibrocystic disease. Affected children are liable to recurrent attacks of wheezy bronchitis. (See chapter on Cough.)

## PATHOLOGY

Wheezing in the chest results from partial obstruction of the bronchi and bronchioles leading to a narrowing of their lumens. Pathological causes of this narrowing can be divided into three groups.

(1) Causes within the lumen: Excessive production of sticky mucus in asthma, bronchitis and fibrocystic disease; foreign bodies of small size will cause a partial obstruction.

(2) Causes within the walls of the bronchioles or bronchi: Spasm of the bronchial muscles in asthma and bronchitis; swelling of bronchial mucous membrane in infective and allergic conditions.

(3) Causes outside the bronchi: pressure from mediastinal glands and other masses.

## DIAGNOSIS

The most frequent causes of wheezing in children are asthma and acute bronchitis which account for 99 per cent of all 'acute wheezy chests'.

An accurate differential diagnosis can usually be made clinically, although at times the distinction between asthma and wheezy bronchitis may be difficult. In practice this distinction is largely academic because the management is similar. The importance lies in discussing the probable prognosis with the parents.

It is important always to bear in mind the rare causes such as inhaled foreign bodies, pressure from enlarged mediastinal glands and fibrocystic disease, for although they are responsible for only 1 per cent of wheezing attacks their treatment is entirely different.

*Type of Wheezing*

In wheezy bronchitis and asthma *attacks* of wheezing occur. In both groups the attack may be preceded by a cold or other acute respiratory infection. This preceding infection is invariable in the case of the bronchitic group and occurs in about a third of attacks of asthma. Where the wheezing results from inhaled foreign bodies or pressure from mediastinal glands the wheezing tends to be *continuous* and worse when the child cries, gets excited or has a cold. Unilateral wheezing is most likely to be due to inhaled foreign bodies or mediastinal swellings.

*History*

In children with asthma there is very often a family history of allergy. Hay fever, eczema and asthma figure prominently in close relatives. It must be remembered, however, that these are common conditions affecting an appreciable proportion of the population. A history of eczema in the affected child is in favour of the diagnosis of true asthma.

*Differential Diagnosis*

The differentiation of 'asthma' from 'wheezy bronchitis' becomes easier as time goes on. The asthmatic tends to go on suffering from recurring attacks of wheezing which, while sometimes associated with a preceding respiratory infection, very often occur for no apparent reason. This recurrent and paroxysmal nature and the association with a family history of allergy are the chief clinical features in deciding on the diagnosis of 'asthma'. This label, however, should be applied only after much consideration and only

when one feels really confident of the diagnosis because of the anxiety its application causes parents.

*Investigations*

In persistent and recurrent attacks a radiograph of the chest is necessary to exclude causes such as mediastinal swellings and local pulmonary lesions.

The tuberculin skin test is not necessary as a routine but should be carried out when enlarged mediastinal glands are found.

Skin testing is often recommended to try to discover possible allergens to which the child may be sensitive. It is the opinion of many physicians that these skin tests are of no real practical value.

## SIGNIFICANCE

*Is the Symptom Present?*

Wheezing is generally obvious when present and there is little difficulty in hearing it. Wheezy children are often little affected by the attacks they suffer.

*What are the Causes?*

'All that wheezes is not asthma.' It is wrong to apply the term asthma to other than a definite and small group of wheezy children with recurrent and paroxysmal attacks of wheezing who have a definite family history of allergic conditions. The majority of wheezy children represent a large group with sensitive respiratory tracts that react to infections of the lower respiratory tract with bronchospasm and wheezing.

Beware of persistent and unilateral wheezes. They denote partial obstruction of the lower airways due to mechanical causes such as inhaled foreign bodies and pressure from mediastinal swellings.

*What to Do?*

An expectant and observant policy is justifiable. Attacks are most frequent between 3 and 8 years of age and in both asthma and acute wheezy bronchitis there is a very definite tendency for attacks to become much less frequent after the age of 8 years.

## SPECIFIC CONDITIONS

### ACUTE WHEEZY BRONCHITIS

The great majority (85 per cent) of acute wheezy chests follow upon a preceding respiratory tract infection and it is presumed that they

represent attacks of acute bronchitis. Boys are affected much more often than girls.

The periods when children are most liable to these attacks are in the first year of life and on starting school. In this group hereditary influences are not very marked and there is no frequent association with eczema.

*Clinical Features*

The onset is with a running nose and fever and a gradually increasing tight cough. Breathing becomes difficult and the child increasingly distressed.

There is audible wheezing even without auscultation of the chest. This is chiefly in the expiratory phase and widespread rhonchi and rales will be heard.

The attacks vary in duration. On an average an improvement begins within 48 hours, and this is often unrelated to any specific treatment.

*Course*

Recurrent attacks are a feature and occur in about a third of children affected. A susceptible child may suffer a wheezy chest with each cold that he gets. However, there is a very definite tendency for these children with recurrent attacks to outgrow them at the age of 7–9 years. By the age of 12 years only 10 per cent of these children are still having attacks, and many of these are not severe.

*Management*

Management depends on an understanding of the cause and on the appreciation that although these attacks are presumably infective, many will settle with the use of simple antispasmodic drugs.

## ASTHMA

The diagnosis of asthma in children should really be one of exclusion. The label should not be attached to a child indiscriminately because of the unnecessary anxiety caused to the parents.

Asthma may be diagnosed when acute attacks of wheezing and breathlessness recur paroxysmally and unassociated with acute respiratory infections or other chest conditions.

With this somewhat vague and rather exclusory definition it is found that the incidence of asthma in childhood is about a tenth that of wheezy bronchitis. In spite of this apparently low incidence it

is a fact that between a third and a half of all asthmatics begin their attacks in childhood.

In childhood boys are more often affected than girls (this is not so in adults where the sex incidence is more equal).

*Clinical Features*

The attacks are sudden, often starting at night. The child suddenly awakens, sits up, holds the side of the bed and fights for breath. There is obvious wheezing and the difficulty is with expiration, the chest being held in a position of full inspiration. Cough is generally present and the sputum is very sticky.

The duration of each attack is variable. With suitable therapy it may be cut short to an hour or two or on the other hand it may go on for many days. The frequency also varies. It is very intimately associated with the emotional situation in the household.

*Course*

The great majority of children with asthma either cease to suffer attacks or improve greatly. This fact is apparent only from a long-term follow-up, and from the reports published between 90 and 75 per cent cease to suffer from major attacks. This improvement occurs from 7 to 12 years of age in most children.

*Management*

The management of children with asthma is a problem, but the approach should be to appreciate the probable satisfactory outcome in the majority. The management of the acute attacks should be simple and effective. Each attack should be treated on its merits with antispasmodics, antibiotics and corticosteroids as indicated. To this non-specific approach must be added a consideration of the various emotional and personal problems of the individual child and family.

TABULATED SUMMARY

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Wheezy bronchitis | Recurrent; sudden onset, variable general upset; follows upper respiratory tract infections; diffuse rhonchi | Clinical | (1) Broncho-dilators (2) Anti-biotics where indicated | Recurrent but tendency to natural improvement |

| *Disease* | *Features* | *Diagnosis* | *Management* | *Course* |
|---|---|---|---|---|
| Asthma | Recurrent; sudden onset; paroxysmal; variable general upset; diffuse rhonchi | (1) Clinical<br>(2) Chest radiograph | (1) Parents as well as child<br>(2) Broncho-dilators<br>(3) Steroids in certain cases | Recurrent but definite tendency to natural improvement |
| Inhaled foreign bodies | History of initial incident; persistent wheeze that may be unilateral | (1) Clinical<br>(2) Chest radiograph (not all inhaled foreign bodies are radio-opaque) | Bronchoscopy if suspected | Permanent residual damage if not treated |
| Enlarged mediastinal glands | Persistent wheeze; worse with respiratory infections | (1) Chest radiograph<br>(2) Tuber-culin skin test | Look for primary cause | Depends on primary disease, for instance tuberculosis, new growths; sarcoidosis |
| Fibrocystic disease | Recurrent chest infections with sputum; steatorrhoea and undigested proteins in stools; non-thriving | (1) Stools<br>(2) Duodenal juice<br>(3) Sweat (excess NaCl) | Long-term antibiotics | Poor prog-nosis; few live to adolescence |

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